# LIFESTYLE LESSONS

## A Guide to Diabetes Care

HV2332
L626
1990

Digitized by the Internet Archive
in 2015

https://archive.org/details/lifestylelessons00unse

# LIFESTYLE LESSONS

## A Guide to Diabetes Care

[illegible]
732
L626
1990

Provided as an educational service by

Pfizer **Roerig**

# LIFESTYLE LESSONS TABLE OF CONTENTS

**Introduction** ........ 1
Alan J. Garber, MD, PhD

**Nutrition and the NIDDM Patient** ........ 5
Marion J. Franz, RD, MS, CDE
International Diabetes Center
Minneapolis, MN

**The Role of Exercise in Type II Diabetes** ........ 23
Stephen H. Schneider, MD
Associate Professor of Medicine
Division of Endocrinology, Metabolism, and Nutrition
UMDNJ—Robert Wood Johnson Medical School
New Brunswick, NJ

**Health Education** ........ 55
Helen Mellett, RN, Dr PH
Adult Nurse Practitioner
Assistant Professor of Nursing
Columbia School of Nursing
New York, NY

**Diabetes Education and Behavior Modification** ........ 61
Jane Darling, RN, ANP, CDE
Diabetes Education Coordinator
Mount Sinai Hospital
Hartford, CT

**Drug Therapy for Diabetes** ........ 77
Alan J. Garber, MD, PhD
Professor of Medicine, Biochemistry, and Cell Biology
Baylor College of Medicine
Chief, Diabetes/Metabolism Unit
The Methodist Hospital
Houston, TX

# LIFESTYLE LESSONS TABLE OF CONTENTS

**Self-Monitoring and Daily Management** ........ 81
Ann Marie Cirkot, RN, BSN, CDE
Community Health Care Plan
Long Wharf Health Center
New Haven, CT

**Foot Care** ........ 93
Ann Marie Cirkot, RN, BSN, CDE
Jane Darling, RN, ANP, CDE

**Patient Information**

This book contains a number of pages that can be copied and given to patients (7, 8, 9, 11, 12, 13, 14, 16, 17, 38, 39, 40, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 79, 90, 94, 95).

# Introduction

**Alan J. Garber, MD, PhD**
**Professor of Medicine, Biochemistry,**
**and Cell Biology**
**Baylor College of Medicine**
**Chief, Diabetes/Metabolism Unit**
**The Methodist Hospital**
**Houston, TX**

Regardless of a patient's age at onset, diabetes mellitus is a life-threatening disease that kills insidiously, day by day. It shortens life span, impairs lifestyle, and requires constant daily—even minute by minute—attention for control. When such careful attention becomes part of a management regimen, a patient may well enjoy a healthy, full life, with only modest restrictions. Today, it is clear that such success is beyond the reach of doctors alone. Instead, it depends on teamwork among health-care professionals, including well-trained patient educators. This manual outlines a diabetes education program for patients of any age—with either severe or mild diabetes.

In diabetes mellitus, insulin regulation no longer controls the metabolism of carbohydrates, proteins, and fats. As a result, the autoregulation of these metabolic fuels and structural components of the body becomes disordered or ceases altogether. When this occurs, the patient is betrayed by his own body's failure.

The traditional goal of diabetes management is to make appropriate modifications in the patient's lifestyle as a way of compensating for this failure of autoregulation. Nevertheless, adaptive lifestyle responses alone are seldom enough: they produce only an approximation of the glycemic control of an intact pancreatic-hepatic-peripheral tissue axis. In order to achieve significant levels of diabetic control, major behavioral adaptations and conscious, anticipatory planning are essential. Indeed, medical strategies for intensified diabetes management depend in part upon careful planning and on the adaptive changes that enable pharmacologic preparations to approximate natural patterns of glycemic control. This process depends, in turn, on effective diabetes educators, for these vital professionals must teach patients to recognize patterns and manage adaptive responses. Only through effective education can patients develop the motivation required to achieve this level of diabetes control.

## Education: The Basis of Success

If diabetes management can be seen as an effort to substitute rational data gathering and behavior for an autoregulatory failure of the patient's endocrine-metabolic axis, it becomes apparent that education is central to successful diabetes management. In fact, diabetes management is really no more than management by patients themselves. The job of the diabetes care team is to serve as coach, coordinator, and even cheerleader to help the patient develop rational, adaptive behaviors that can substitute, at least in part, for internal autoregulation.

The only outpatient diabetes management that counts is self-management. This statement applies to all forms of diabetes mellitus, including mild cases of transient hyperglycemia. Only the degree of behavioral change necessary for self-management distinguishes the varying severities of diabetes mellitus. The role of those who treat diabetes mellitus must be reevaluated in light of this construct.

Authoritarian educational approaches are unwise and often counterproductive for patients with diabetes mellitus. Success comes most often to educators who can negotiate medical management plans that fit the lifestyle, desires, and necessities of each individual patient. Careful assessment of these factors must precede the actual management plan. This entire process should be focused on reducing the risk of acute and chronic complications of the disease.

### The Management Plan

In formulating a management plan, aggressive glycemic control is an essential first step. But balancing the variables of diet, exercise, and pharmacologic agents must take into account a series of factors. All have to be negotiated based on the patient's lifestyle and the physician's perception of which therapeutic strategies best answer the patient's needs.

Obese patients who have non-insulin-dependent diabetes mellitus (Type II) may best be managed by diet and exercise alone. However, if this fails to produce good glycemic control it is not necessarily the patient's fault. Nor is it solely a failure of the treatment team. That fact should be clear to all because failures of this kind are common in the management of patients with diabetes mellitus and are due to many variables. Reliance on lifestyle strategies alone for unduly long periods of time can prolong the hyperglycemic burden and the attendant risk of complications.

Prescriptions for pharmacologic regimens that impose too many changes in the patient's lifestyle often result in noncompliance. Similarly, the businessman with hectic schedules and erratic meal patterns is not best managed with intermediate-acting insulin alone because rigid meal patterns are a necessary part of such therapy. This insulin program will soon become burdensome to the patient and is likely to produce noncompliance. The patient will begin to adjust his insulin dosages capriciously—or eliminate them entirely—to avoid hypoglycemic reactions in the middle of the business day.

Oral sulfonylurias, such as the second-generation agents that can be given once daily, are particularly useful in these patients. Because these rapid-acting agents, such as glipizide, institute insulin release only in response to meals, they have additional benefits for active diabetics as well as for sedentary elderly ones. (In both of these patient populations, insulin uptake may be erratic.)

In addition to their duties as negotiators, diabetes educators must often mediate between the doctor and the patient. A regimen may not meet the patient's needs, and the patient may be unable, unwilling, or insufficiently knowledgeable to discuss its impracticality. When this happens, the educator's careful assessment of lifestyle factors can provide the information that will produce a workable diabetes program. For this reason, the most successful management plans result from team effort. Actually, the best plans are established when educators and dietitians assess the patient's needs and communicate them to the doctor before recommendations for management are made. Too often, patients are intimidated by physicians, and are unwilling to discuss their own needs. The consequence is that educators and dietitians become patients' true confidants; they become conduits for delivering information between patients and their physicians. Thus, the team approach to diabetes is justified not merely because of the specialized abilities of its members, but also because the team as a whole is far more effective than any of its various parts.

### Important Relationships

The educator should be a trained professional able to teach patients the techniques that make self-management possible. The educator also must acquire expertise with the various devices and instruments that can augment self-management. These range from traditional injection techniques to pumps and new insulin injection systems. They also include blood and urine self-monitoring devices. The educator is charged with instilling in the patient specific manual skills as well as an understanding of their significance. Only when patients are taught the "how and why," can they act rationally to compensate for endogenous autoregulation of the endocrine-metabolic milieu. Of even greater significance is the role educators can play as counselors and motivators. This is possible because patients perceive the educator as a less threatening, more nurturing figure than the physician. As a result, effective diabetes educators often can approach issues of behavior and compliance more positively and less emotionally. Because their relationship is basic to success in diabetes management, the interaction between patient and educator must be a continuing process. Otherwise, it may be impossible to encourage the behavioral modifications that lead to successful adaptation to the diabetic state. The nature of the educator's role is so crucial that it may be wise for this professional to lead the team's contact with diabetic patients. In this way, the problems reprimanding the patient and causing guilt, and the maladaptive, hard-to-reverse behavior that these problems inspire, can be avoided.

While the educator initiates the patient into diabetes management, and should be a sustaining force, the relationship between the patient and dietitian is no less vital. It is apparent that initial dietary regimens often are not suited to the individual lifestyles of patients. Such arbitrary dietary regimens are unlikely to be followed. Prescriptions that strive to achieve rapid weight loss by dramatically cutting caloric intake will soon be ignored by patients for whom the change is too radical in the context of accustomed behaviors. Only by having the dietitian serve as the initial contact in assessing dietary strategy can habitual behavior be modified sufficiently to allow patients to follow dietary programs successfully. Well-tailored regimens can prevent rebellious, counterproductive behavior.

Dietitians also can improve the relationship between physician and patient. This is true because frequent contact with the dietitian transfers the physician's job of admonishing the patient about failures of weight loss to a surrogate who can take a less authoritarian approach to lapses in compliance. As a consequence, relationships between the team and patients remain more cooperative.

Progress in diabetes management has been enhanced by technological advances in the 1970s and 1980s. During this time, it has become apparent that management teams are the most successful means of enlisting and amplifying patients' participation in their own care. In fact, such teams are absolutely essential in implementing technology.

The new frontier in diabetes management lies in helping patients make psychological and attitudinal adjustments to their disease. Many people are diabetic precisely because of counterproductive, often pathologic patterns of eating and exercise that predate onset by years and even decades. Reversal of such patterns is a formidable challenge that we are only now beginning to meet. All of us hope that further technological advances will obviate the need for intensive psychological intervention, but those advances are on the distant horizon. Totally implantable closed-loop systems for delivering insulin and pancreatic islet transplants are not likely to become routine in the near future. Because the latter are the only mechanisms that could circumvent the need for readjusting patients' counterproductive attitudes and behaviors, strategies that currently focus on such readjustments will not be discarded in the foreseeable future. Indeed, the future depends upon their successful implementation.

# Nutrition and the NIDDM Patient

**Marion J. Franz, RD, MS, CDE**
**International Diabetes Center**
**Minneapolis, MN**

In reviewing the role of nutrition in managing non-insulin-dependent diabetes mellitus (NIDDM) three questions need to be addressed:

- What are the current nutritional recommendations for NIDDM?
- What educational system will promote understanding as well as positive behavioral changes?
- Which educational tools reflect current nutritional recommendations and can be used within the educational system?

By answering these questions, the diabetes educator can develop an effective approach to helping patients alter their meal planning.

## Nutritional Recommendations

The American Diabetes Association's (ADA) *Nutritional Recommendations and Principles for Individuals with Diabetes Mellitus* (1986) provide the basis for nutritional management of diabetes.[1] Table 1 summarizes the history of changes in guidelines for macronutrient content.[2] It is important to note that the latest nutritional recommendations for people with diabetes are similar to those of the American Heart Association,[3] the Step-One diet of the National Cholesterol Education Program,[4] the National Cancer Institute,[5] the American Cancer Society,[6] and the U.S. Dietary Guidelines.[7]

## Calories

In setting total calories for a meal plan, the aim must be to help patients achieve and maintain desirable body weight (DBW).[1] Although reducing to achieve a DBW is commonly listed as the first goal of nutritional management, "reasonable" body weight is usually a more realistic way of stating the goal. Reasonable body weight is defined both by short- and long-term goals the patient and nutrition counselor feel are achievable. These may be quite different from "desirable" weights.

Two terms used, often interchangeably, to describe excess body weight are "obesity" and "overweight." Obesity is defined as an excessive amount of adipose tissue, regardless of a patient's

Table 1

**Percentage of Calories from Carbohydrate, Protein, and Fat**

| Date | CHO | Protein | Fat |
|---|---|---|---|
| <1921 | Starvation diets | | |
| 1921 | 20 | 10 | 70 |
| 1950 | 40 | 20 | 40 |
| 1979 | 50-60 | 12-20 | 30-35 |
| 1986 | Up to 55-60 | 12-20 | 25-30 |

From Franz.[2]

actual weight, and is a risk factor for NIDDM. Another risk factor associated with obesity is abdominal obesity versus gynoid or peripheral distribution of fat.[8] Abdominal obesity is associated with a greater risk for NIDDM and coronary heart disease as well as for higher glucose levels and insulin resistance.

The term "overweight" simply indicates excessive heaviness, and may or may not reflect a measure of adipose tissue. In fact, heavily muscled people, who have an overabundance of lean body tissue, can be overweight.

As research continues to uncover reasons why weight loss is so difficult for many patients,[9,10] we may need to shift our therapeutic emphasis from weight loss to weight management, coupled, perhaps, with medication to normalize blood glucose and lipids for patients. Then, weight loss could be considered an added benefit.

If weight reduction is achieved, potential benefits include reduction or elimination of exogenous insulin requirements, an increase in life expectancy, and improved health in general.[10] Not only does weight loss assist with blood glucose control, it also improves blood pressure and blood lipid (cholesterol and triglyceride) levels. The mortality rate for patients who are 20 percent to 30 percent above ideal body weight is 2.5 to 3.3 times higher than for those of normal weight. The mortality rate is 5.2 to 7.9 times higher among diabetes patients whose body weights are 40 percent or more above the ideal.[11]

Many overweight NIDDM patients do not ingest excessive amounts of food.[10] Still, they find it difficult to achieve and maintain proper body weights. Several factors can contribute to this: genetic predisposition,[12] a defective regulatory system that maintains an elevated body weight,[13] altered energy efficiency,[10] reduced thermogenesis,[14] or a support system that does not establish weight loss as a priority. Weight loss is particularly hard for adults over age 55, who have reduced energy requirements due to decreases in the basal metabolic rate, changes in body composition, and decreased physical activity.

The **goals for weight management** are:

- Improved eating habits
- Moderate caloric restriction (500 to 1,000 kcal less per day than diet history) to produce a gradual and sustained weight loss
- Better spacing of meals—especially to avoid large caloric intakes late in the day and evening
- Learning new behaviors and attitudes that make long-term lifestyle changes easier
- Increased physical activity (note: exercise without concomitant restriction of calories rarely results in significant loss)[15]

When used with medical supervision, very low-calorie diets have been shown to lead to significant weight loss and improved metabolic status. Such efforts can even reduce or eliminate the need for oral hypoglycemic agents or insulin.[16] However, very low-calorie diets are not generally recommended for persons who need to lose less than 50 pounds. The American Diabetes Association Task Force on Nutrition cautions that further studies of the prolonged use of such diets are necessary before they can be recommended.[1]

It would be ideal if we could use percentage of body fat as a convenient and accurate means of establishing body weights. Because this often is not possible, Tables 2, 3, and 4 are designed as a guide to body weight and caloric levels. An alternative approach is to use a nutritional history of accustomed daily intake. Patients who eat 500 to 1,000 calories a day less than usual should achieve a gradual weight loss of one to two pounds per week.

The minimum recommended caloric level is

Table 2

### To Estimate Desirable Body Weight (DBW), Use This Formula

**Women:** 100 lb for the first 5 ft of height
5 lb for each additional inch

**Men:** 106 lb for the first 5 ft of height
6 lb for each additional inch

*(Add 10% for large body frame. Subtract 10% for small body frame.)*

From American Diabetes Association and The Americon Dietetic Association.[32]

Table 3

### To Estimate Daily Caloric Requirements, Use This Formula

To determine daily caloric requirements, multiply DBW by the appropriate number of calories.

15 calories per lb of DBW for moderately active men and physically active women.

13 calories per lb of DBW for most women, sedentary men, and adults over age 55.

10 calories per lb of DBW for very obese or very sedentary people, or people who have been on low-calorie diets for long periods of time.

From American Diabetes Associotion and The American Dietetic Associotion.[32]

Table 4

### To Estimate Caloric Requirements for Weight Loss, Use This Formula

The following formula can be used to estimate approximate caloric requirements:

1. Determine desirable body weight (DBW) (See Table 2) __________
2. Multiply DBW by approximate calories needed (See Table 3) ×__________
3. Daily caloric requirement =__________calories
4. To lose 1 lb per week, subtract 500 calories from daily requirement. To lose 2 lb subtract 1,000 calories from daily requirements (To gain, add 500-1,000 calories) –__________

Daily caloric requirement to lose (gain) weight =__________

Adapted from Franz, et al.[33]

**Copy This Page for Patient Information**

Table 5

**Calorie Content Guide**

| Food Group | Serving Size | Calories |
|---|---|---|
| 1. Starch/bread | 1 slice bread, ½ cup pasta, starchy vegetable, potato, ½ to ¾ cup cereal, 4 to 6 crackers | 80 |
| 2. Meat and substitute | 1 oz cooked lean meat (poultry, fish, lean beef, canned ham), ¼ cup cottage cheese | 55 |
| | 1 oz cooked medium fat meat (beef or pork roast, steak, lean ground beef), liver, 1 egg | 75 |
| | 1 oz cooked high fat meat (prime cuts, spare ribs, etc.), 1 oz cheese, sausage, or luncheon meat, 1 hot dog, 1 tablespoon peanut butter | 100 |
| 3. Vegetable (nonstarchy) | ½ cup cooked,<br>1 cup raw | 25 |
| 4. Fruit | ½ cup canned or frozen without sugar,<br>1 medium serving of fresh,<br>1 cup melon or berries,<br>¼ cup dried fruit | 60 |
| 5. Milk | 8 oz skim or low-fat milk<br>8 oz plain low-fat yogurt | 90 |
| 6. Fat | 1 teaspoon margarine,<br>2 teaspoons diet margarine or "lite" mayonnaise,<br>1 teaspoon oil or mayonnaise<br>1 tablespoon salad dressing (French or Italian)<br>2 tablespoons reduced-calorie salad dressing | 45 |

From Monk, et al.[34]

**Copy This Page for Patient Information**

Table 6

**Determining the Meal Plan**

| Food Group | No. of servings that you desire | Calories per serving | Calories |
|---|---|---|---|
| 1. Starch/bread | ________ | × 80 = | ________ |
| 2. Meat and substitute | ________ | × 75 = | ________ |
| 3. Vegetable | ________ | × 25 = | ________ |
| 4. Fruit | ________ | × 60 = | ________ |
| 5. Milk, skim | ________ | × 90 = | ________ |
| 6. Fat | ________ | × 45 = | ________ |
| | | Total Calories | ________ |

From Monk, et al.[34]

Table 7

**Ideal, Borderline High-Risk, and High-Risk Cholesterol Values**

| | Ideal | Borderline high (moderate) risk* | High risk |
|---|---|---|---|
| Total cholesterol (mg/dL) | <200 | 200-239 | >240 |
| Low-density lipoprotein cholesterol (mg/dL) | <130 | 130-159 | |

* Borderline risk becomes high risk if individual has diagnosed heart disease (CHD) or two or more of the following risk factors:

Male gender
Family history of premature CHD
Smoking
Hypertension
Diabetes
Low high-density lipoprotein cholesterol
History of definite peripheral vascular disease
Severe obesity

From Cholesterol Adult Treatment Panel Report.[4]

**Copy This Page for Patient Information**

1,200 calories for women and 1,500 calories for men. These amounts are necessary to ensure that patients' diets include all the necessary vitamins and minerals. Table 5 outlines average caloric levels for the six different food groups. Table 6 can be used to fit an appropriate number of servings from each food group into a diet's total caloric level. To lose more weight than is possible through calorie reduction alone, caloric expenditure through exercise should be increased.

If diet alone is the therapy of choice, the educator and the diabetes patient should agree on a target date for achieving the desired glucose level. If this level has not been achieved by diet alone by the consensual date (usually within one to three months), the patient should be placed on medication, either an oral hypoglycemic agent or insulin.

### Fat and Cholesterol

The ADA's 1986 nutrition guidelines recommend that total dietary fat comprise less than 30 percent of the dietary calories. At the same time, the intake of dietary cholesterol must be less than 300 mg/day.[1] Within these limits, meal plans should be made palatable and the amounts recommended must be reasonable in the context of the overall nutritional prescription. Otherwise, noncompliance is almost certain to result.

Persons with diabetes have a disproportionately high risk of cardiovascular disease. Reducing fat content significantly decreases this risk. Abnormalities of plasma lipids and lipoprotein concentrations are also associated with greater cardiovascular risk. Because of the established favorable effects fat-modified diets have on lipids and lipoprotein concentration, the minimum dietary requirements for persons with diabetes should be comparable to the National Cholesterol Education Program Step-One recommendations.[4] All types of fats—saturated, polyunsaturated, and monounsaturated—should be proportionally reduced. Table 7 lists target serum cholesterol levels, and Table 8 shows guidelines for reducing dietary fat.

### Carbohydrates

The amount of carbohydrates in a diabetes patient's diet can account for up to 55 percent to 60 percent of total caloric intake.[1] However, these recommendations have been controversial, especially in relation to NIDDM.[17] They do not say that a meal plan must contain 55 percent to 60 percent of the calories from carbohydrate, but emphasize that the prescribed percentage is to be individualized. The actual recommended amount will depend on an individual's eating habits and the impact of carbohydrates on blood glucose and lipid levels. In fact, it is very difficult to design a realistic meal plan in which more than half the calories are derived from carbohydrates. This is especially true for lower calorie meal plans. Individualization is necessary if we are to help patients meet the goals of diabetes management.

Whenever possible, patients should choose foods containing fiber, over foods with highly refined carbohydrate. The latter are low in fiber content and fail to provide certain benefits. Water-soluble fiber, which is found in legumes, oats, barley, and some fruits, appears to form a gel within the gastrointestinal tract, thus slowing the absorption of glucose across the intestinal mucosa as well as lowering total cholesterol and LDL-cholesterol. Water-insoluble fiber, found in wheat and corn bran, whole grains, and some vegetables, increases fecal weight and bulk within the gastrointestinal tract, and may help relieve constipation and other gastrointestinal problems.

Estimates are that American adults consume between 10 and 30 g/day of dietary fiber. Men average 19 g and women 13 g. A daily intake as high

Table 8

## Guidelines for Reducing Fat in Your Diet

) Lower total meat consumption. Select the leanest cuts and remove all visible fat before eating.
  - limit meat, fish, poultry, cheese and eggs to 6 oz or less daily (two small servings).
  - increase use of vegetable protein (legumes, beans, peas, nuts, and seeds) in place of animal protein.

) Select poultry (preferably without skin), fish, lean beef, and vegetarian fare.

) Avoid high-fat meats, such as luncheon meats, frankfurters, sausage, and bacon.

) Decrease total fat intake by baking, broiling, roasting, grilling, or boiling foods. Avoid frying. Skim fat from broths, soups, and gravies.

) Drink skim milk.

) Use low-fat or nonfat dairy products such as plain yogurt and part skim-milk cheese. Avoid dairy products containing large amounts of butterfat.

) Use margarine instead of butter. Be sure a liquid oil is listed as the first ingredient on the margarine label.

) Avoid food products having the term "hardened" or "hydrogenated" vegetable oil as a major ingredient.

) Use vegetable oils whenever possible in your baking or cooking and salad dressings.

) Decrease intake of rich desserts.

From Franz, et al.[33]

**Copy This Page for Patient Information**

Table 9

### Guidelines for Adding Fiber to Your Diet

- Increase your fiber intake slowly. Intestinal gas may occur when fiber intake is increased. Generally, this is only a temporary problem and will subside as the body adjusts to a higher fiber diet. Start with small amounts of fiber and increase levels gradually.
- Eat a variety of fiber-rich foods in reasonable amounts. Increase your intake of whole-grain breads and cereals. Include more fruits and vegetables in your diet. Bran cereal is an especially concentrated source of fiber. Fiber should come from food, not fiber supplements.
- Eat carbohydrates that are still in their natural fibrous coatings. Choose brown rice instead of white rice, whole-grain flour instead of white flour; leave skin on fruits and vegetables; eat baked potatoes with skin instead of mashed potatoes.
- Try to drink at least eight glasses of liquids a day. Because fiber attracts water into your intestines, it's important to drink enough liquids when you add fiber to your diet.
- Buy whole-grain bread made from stone-ground flour or 100% whole-wheat or other whole-grain bread. "Brown breads" contain little or no whole grain, just molasses for coloring. Remember, almost all flours are made from wheat, so if the label says "wheat flour," it probably refers to bleached white flour. "Whole grain," "whole wheat," or "whole oats" should be first on the ingredients list.
- Use raw fruits, such as apples with skins, oranges with membranes, or fruits with seeds, such as strawberries, instead of fruit juices. Include more raw or slightly cooked vegetables, such as corn, peas, beans, legumes, and potatoes with the skin. The stems and leaves of salad greens and broccoli are also fibrous.
- Make nutritious high-fiber snack foods. Munch on fresh fruits and vegetables, nuts, and popcorn.
- Watch those calories. If you add extra calories by adding high-fiber foods to your daily diet instead of substituting them for some food choices, you'll probably gain weight. To save calories, trim your intake of high-fat, high-calorie foods such as cream, gravies, sauces, and salad dressings. Eat breads and vegetables without margarine or butter; use noncaloric products (such as Pam® vegetable spray) for greasing pans, or use nonstick pans.
- Look for dietary fiber content on food labels.
- Choose lean meats and eat only 4 to 6 oz per day.
- Limit fat and oil.

From Franz.[35]

**Copy This Page for Patient Information**

Table 10

**Average Dietary Fiber Content of Food Groups**

| | | |
|---|---|---|
| Cereals: | Oatmeal, Wheaties, Shredded Wheat, Bran Flakes, Grapenuts, Corn Bran, Chex (average ½ cup) | 3 g |
| | 100% Bran, All Bran, Bran Buds, Fiber One, (⅓ to ½ cup) | 8 g |
| Starchy Vegetables and Grains: | Corn, potatoes, brown rice, bulgur, peas (⅓ to ½ cup) | 3 g |
| Dried Beans and Peas, Lentils: | (⅓ cup) | 4 to 5 g |
| Breads: | Whole-grain breads and crackers (1 slice or 1 oz) | 2 g |
| Vegetables: | Raw or cooked<br>½ to ¾ cup cooked<br>1 to 2 cups raw | 2 g |
| Fruits: | Fresh or canned or frozen<br>½ cup or one medium fresh | 2 g |
| | Dried fruits, ¼ cup | 3 g |

From Monk, et al.[34]

**Copy This Page for Patient Information**

Table 11

## Guidelines for Reducing Salt in Your Diet

- Use the salt shaker less often, if at all. Don't set it on the table.
- Cook with less salt.
- Limit your use of commercial snack foods, "convenience" processed foods, and fast foods. (Home-cooked is still best.)
- Read package labels. The amount of sodium will be listed on product labels that carry nutritional labeling.
- Carefully choose your condiments. High-salt condiments include soy sauce, steak sauce, catsup, and seasoned salts.
- These seasonings contain sodium and are best limited or avoided:

Celery salt
Meat/vegetable extracts

Seasoned salt
Meat tenderizers

Garlic salt/Onion salt
Commercial bouillon (cubes or granules)

Cooking wine
Catsup

Chili sauce
Prepared mustard

Steak sauce
Table or seasoned salt

Barbecue sauce
Olives, pickles

Soy sauce
Monosodium glutamate

Worcestershire sauce
Brine

From Monk, et al.[34]

**Copy This Page for Patient Information**

as 40 g of fiber (25 g/1,000 kcal) may be beneficial. For most individuals, the goal should be to double gradually current intake up to 40 g of fiber per day. Fiber supplements are not encouraged.[18]

Studies suggest that dietary fiber is effective in controlling blood glucose and reducing plasma cholesterol, especially when used in very high-carbohydrate diets (greater than 50 percent of calories).[19] Nevertheless, the benefits of fiber on metabolic control have been questioned.[20] Realistically, it is difficult to increase fiber to 40 g/day. But from a nutritional standpoint, it does seem prudent to encourage patients to increase dietary fiber, regardless of its effect on blood glucose. Table 9 has guidelines for adding fiber to the diet. Educators can use these guidelines when counseling patients. Table 10 lists average grams per serving of dietary fiber in various food groups.

Modest amounts of sucrose and other refined sugar may be acceptable for some patients.[1,15] Their use is contingent on the effects on blood glucose and lipids as well as maintenance of body weight. The statement concerning sucrose use is not intended to encourage the use of foods high in refined sugars, but to help patients use these foods correctly. If they are included in the meal plan, portions should be small and patients should be taught how to make correct substitutions for them. Such foods are best consumed at meal times, when they appear to be absorbed more slowly. Caution in using significant amounts of sugar is good nutritional advice for everyone.

While the role of sucrose is also controversial,[21-23] *The National Institutes of Health Consensus Statement on Diet and Exercise in NIDDM*[15] declares that sucrose is acceptable as a taste additive in mixed meals (up to five percent of carbohydrate calorie intake) for lean patients who do not have carbohydrate-aggravated hyperlipidemia. Moreover, to tell most people not to eat sugar is to give advice that cannot be followed. Because of its ubiquitous role as a food additive, sugar is difficult to avoid.

### Protein

The recommended dietary allowance (RDA) of protein for adults is 0.8 g/kg of body weight.[24] The elderly, however, may require slightly more than this, because they frequently require repletion of body protein during convalescence from illness. Using 1 g/kg, a 130-pound woman requires only 59 g of protein daily; a 170-pound man needs 77 g. In general, Americans consume two to three times the RDA of protein. The usual recommendation for protein (for people with diabetes) is 12 percent to 20 percent of total calories.

### Alternative Sweeteners

The use of alternative sweeteners, both nutritive and non-nutritive, is thought to be acceptable in the management of diabetes. Even so, the use of these substances should be evaluated in the context of a diet's overall nutritional value.[25,26] Patients should be warned about the caloric content of such nutritive sweeteners as fructose and sorbitol. They should be urged to use a variety of sweeteners to dilute any potential risk.

### Salt

The American Heart Association says sodium intake should not exceed 3,000 mg/day. The principal concern is over salt's effect on hypertension. People with diabetes are frequently hypertensive, and control of this condition must be one of the first steps in controlling the micro- and macrovascular complications associated with diabetes. Caution should be taken with patients who have problems with fluid volume or hypotension; salt intake may be beneficial for these individuals.[1] Table 11 sets guidelines for reducing salt in the diet.

Table 12

## Using Information on Food Labels

Food labels can assist you in deciding which foods are appropriate to use in meal planning and how to use them correctly. Three general guidelines can be helpful.

1. *Descriptive terms used on labels.* Some food labeling terms are well regulated and clearly specify how the term can be used. Terms regulated by FDA are: low-calorie, reduced calorie, sodium-free, very low sodium, low sodium, reduced sodium, enriched, fortified, imitation. The following terms are not regulated or if they are regulated, they can be misleading to the consumer: light or lite, sugar-free, sugarless, low-salt, no cholesterol, natural, organic.

   There are a large number of food products such as dietetic candies, cookies, cakes, ice cream, and pudding mixes that usually contain as many or more calories, usually from added fat, than the product they were designed to replace. To evaluate products labeled as "diet," "dietetic," or "dietary" begin by looking at the total number of calories provided from one serving. (Look closely at what is considered a serving size.) If the product contains less than 20 calories per serving, a serving may be used as a "free food," one per meal or snack. If a serving contributes more than 20 calories per serving, it is not a free food and must be counted in the meal plan regardless of how it is labeled.

2. *Ingredient listing.* Ingredients in a product are listed on the label in descending order of predominance by weight.

3. *Nutritional labeling.* Nutritional labeling is required for any food product for which special nutritive claims are made or to which extra nutrients are added. On other products it is voluntary. Any company that offers nutritional labeling must follow a standard format. The label must state serving size and number of servings per container; calories, carbohydrates, protein, fat, sodium, and percentages of leader vitamins and minerals per serving. Cholesterol, and saturated and polyunsaturated amounts are optional.

From Monk and Franz.[36]

**Copy This Page for Patient Information**

Table 13

## Eating Away from Home

| Choose or ask for | Instead of |
|---|---|
| Salads with dressing on the side | Ready dressed salads |
| Italian, oil and vinegar, French dressing | Roquefort, Thousand Island, mayonnaise-based dressings |
| Baked, roasted, broiled, grilled fish, chicken, meats | Fried meats, fish, chicken |
| Broth-based vegetable soups | Creamed soups |
| Baked or steamed potatoes, rice, noodles | Fried, creamed, scalloped potatoes or pastas |
| Plain vegetables with lemon | Vegetables in cream or cheese sauce |
| Skim, buttermilk, or 1% milk | Whole or 2% milk |
| *Au jus* meats | Gravies or sauces |
| Fresh fruit or fruit juice | Fruit pies, rich cakes |
| Sherbet, frozen yogurt | Ice cream desserts |
| A "people bag" for excess meat, fish, poultry | Eating your entire serving |

From Franz, et al.[33]

**Copy This Page for Patient Information**

### Alcohol

Alcohol use is not encouraged for diabetes patients because of their already increased risk for other medical problems, including hypoglycemia, glycemic control, obesity, and hyperlipidemia. Especially for persons with NIDDM, alcohol consumption intensifies these risks.

If a patient chooses to consume alcohol, it should be done in moderation and within prescribed limits. For diabetics who require exogenous insulin, occasional use of two equivalents of an alcoholic beverage can be consumed in addition to the meal plan. Because of the danger of alcohol-induced hypoglycemia, and because alcohol does not require insulin to be metabolized, food should be not omitted to accommodate its use. Occasional use is defined as not more than two equivalents once or twice a week. One equivalent (½ ounce of pure ethanol) is equal to the amount of alcohol contained in 1.5 ounces of hard liquor, 4 ounces of wine, or 12 ounces of beer. Dry wines and light beers contain less carbohydrate than sweet wines or regular beer and are better choices. Such sweet mixes as tonic, soda pop, or fruit juice can contain significant amounts of carbohydrate or sugar and should be avoided. Alcoholic beverages should always be consumed with food.[1]

Because alcohol is high in calories, its use may be a significant factor where weight is concerned. This is more likely to be a problem for NIDDM patients. Fat exchanges are used in calculating alcohol intake in the meal plans of these patients because alcohol is metabolized in a manner similar to fat (one alcohol equivalent equals two fat exchanges).

### Vitamins and Minerals

There is no evidence that persons with diabetes have unique needs that demand vitamin or mineral supplementation. In fact, the same vitamin and mineral recommendations for the general public also apply to diabetes patients. Those on very low-calorie diets (less than 1,000 kcal) may require supplements.

### Systems for Education

It is essential that education as well as the meal plan be individualized for persons with diabetes. Plans for education must begin immediately after diagnosis. Initial planning requires a coordinated team effort by the physician, dietitian, nurse, and patient. Sometimes, it is necessary to seek family counseling to help patients through the adjustment process in accepting the realities of diabetes. In fact, continuing education is essential if behavioral changes are to be implemented permanently. For such chronic diseases as diabetes, periodic follow-up should occur regularly. For adults, counseling on nutrition should be done at approximately six-month intervals so that subtle lifestyle changes can be noted and appropriate changes made in meal planning.[27] The goal of nutritional education and counseling is to encourage positive behavioral changes, not simply to transfer knowledge. Changes in behavior, particularly when they are related to dietary habits, are not easy to achieve. Effective education requires teachers and counselors who can facilitate positive change.

An excellent source for developing effective educational approaches is *Goals for Diabetes Education*.[27] This book was written as an aid to planning and evaluating education and counseling programs for diabetes patients. Its focus is on education as it occurs at different phases and as a continuous process, because patients' needs change as they adjust to the reality of their disease and embark on the continuing process of developing self-management skills. At diagnosis, education begins on a survival level. But once the adjustment process has begun, the patient is ready to assimilate detailed knowledge in continuing increments. Unfortunately, because of learning limitations or lack of available education and counseling, some never go beyond the basic level of education.

Too often, patients are overwhelmed at diagnosis with an overload of information. And while most diabetes educators realize how much knowledge and how many skills are needed to manage the disease successfully, they overcompensate by trying to teach everything during this initial encounter. Perhaps this is a response to concern that they will not see the patient again. Whatever the reason, the anxious patient absorbs very little and often leaves confused about what to do.

As experience is gained in living with diabetes and the objectives of the initial phase are achieved, the patient is ready to move to the next phase of education. This phase focuses on increasing the knowledge, skills, and flexibility that must be incorporated into a patient's lifestyle if diabetes is to be managed successfully. Tables 12 and 13 can be used to add flexibility to meal planning.

### Educational Tools

No single educational device works in every situation. For each phase of education, different materials may be necessary: simple tools for beginning instruction and more complex ones as the instructional process advances. It must be recognized that some patients do not learn well by reading. For these people, oral instruction using posters, models, demonstrations, and audiovisual programs is useful.

To promote understanding of and adherence to the nutrition goals of diabetes management, the counselor must provide more than the "do's and don'ts" of selecting specific foods and menu information. Beginning a discussion by explaining why diet is important, instead of detailing steps for management, may provide a foundation of basic information about nutrition as it relates to diabetes. The patient should understand basic nutritional principles and their connection with diabetes, and receive an individualized meal plan based on goals of mutual consent.

Several approaches to meal planning are available for counselors who teach diabetes nutritional guidelines.[28] The counselor should be familiar with the various approaches to meal planning and use the one that is most appropriate for the individual patient.

*Health Food Choices* and *Exchange Lists for Meal Planning* are two popular educational tools developed by the American Diabetes Association.[29] Several low-literacy materials[30,31] are also available for use. Using an individually designed meal plan and any of these planning approaches allows for a variety of food choices and ensures an appropriate daily intake of calories, carbohydrate, protein, and fat, as well as necessary vitamins and minerals. These teaching tools reflect the principles of nutrition for diabetes as they are currently understood. *A Guide for Professionals: Diabetes Nutrition and Meal Planning* is a facilitator's manual that can assist professionals with nutritional education and counseling.[32]

### Conclusion

The patient's progress can be monitored with a variety of tools, including daily (24-hour) food diary, or a diary that records specific behaviors. Achievement of treatment objectives, including target weight, blood pressure, and blood glucose levels are valuable. Normalization of lipid levels and glycosylated hemoglobin concentrations is also important. However, it is important to separate adherence from metabolic control. It cannot be assumed that patients in good metabolic control are always compliant, nor that those in poor control are noncompliant. Many factors other than adherence, such as the adequacy of a treatment regimen, may disrupt metabolic control.

For positive changes to occur, family members and "significant others" must be integrated into the educational program. Family members should be encouraged to follow nutritional recommendations similar to those for the patient. This will encourage the patient to comply with a prescribed regimen.

Nutrition is a changing field. For diabetes patients to incorporate nutritional recommendations and principles into their management programs and lifestyles, initial and continuing education, as well as using appropriate educational tools, is essential.

## References

1. American Diabetes Association Task Force on Nutrition and Exchange Lists: Nutritional recommendations and principles for individuals with diabetes mellitus: 1986. *Diabetes Care* 1987;10:26-132.

2. Franz, MJ: Diabetes and nutrition: State of the science and the art. *Top Clin Nutr* 1988;3:1-16.

3. American Heart Association Committee on Nutrition: *Dietary and Coronary Heart Disease*. New York, American Heart Association, 1965 (revised 1968, 1978, 1986).

4. Cholesterol Adult Treatment Panel Report: Report of the national cholesterol education expert panel on detection, evaluation, and treatment of high blood cholesterol in adults. *Arch Intern Med* 1988;148:36-69.

5. Committee on Diet, Nutrition, and Cancer: *Diet, Nutrition, and Cancer*. Washington, DC, National Research Council, 1982.

6. American Cancer Society: *Nutrition and Cancer: Cause and Prevention*. New York, American Cancer Society, 1984, pp 121-126.

7. *Nutrition and Your Health: Dietary Guidelines for Americans*, ed 2. US Department of Agriculture and US Department of Health and Human Services, Home and Garden Bulletin No. 232, 1985.

8. Kissebah A, Vydelingun N, Murray RW, et al: Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab* 1982;54:254-260.

9. Beebe CA (ed): Obesity management in people with diabetes. *Diabetes Spectrum* 1988;1:17-56.

10. Hanson BC: Dietary considerations for obese diabetic subjects. *Diabetes Care* 1988;11:183-188.

11. Blackburn GL, Read JL: Benefits of reducing revisited. *Postgrad Med J* 1984;60:13-18.

12. Stunkard AJ, Foch TT, Hrubec Z: A twin study of human obesity. *JAMA* 1986;256:51-54.

13. Keen H, Thomas BJ, Jarrett RJ, et al: Nutrient intake, adiposity, and diabetes in man. *Br Med J* 1979;1:655-658.

14. Ravussin E, Lillioja S, Knowler WC, et al: Reduced rate of energy expenditure as risk factor for body-weight gain. *N Engl J Med* 1988;318: 467-474.

15. National Institutes of Health: Consensus development conference on diet and exercise in non-insulin-dependent diabetes mellitus. *Diabetes Care* 1987;10:639-644.

16. Henry RR, Wiest-Kent TA, Scheaffer L, et al: Metabolic consequences of very-low-calorie diet therapy in non-insulin-dependent diabetic and nondiabetic subjects. *Diabetes* 1986;35:155-164.

17. Hollenbeck CB, Swislocki ALM, Coulston AM: Persistence of the deleterious metabolic effects of high-carbohydrate, low-fat diets in patients with non-insulin-dependent diabetes mellitus (NIDDM). *Diabetes* 1987;36:12a.

18. Vinik AI, Jenkins DJA: Dietary fiber in management of diabetes. *Diabetes Care* 1988;11:160-173.

19. Anderson JW, Bryant CA: Dietary fiber: Diabetes and obesity. *Am J Gastroenterol* 1986;8:898-906.

20. Hollenbeck CB, Coulston AM, Reaven GM: To what extent does increased dietary fiber improve glucose and lipid metabolism in patients with non-

insulin-dependent diabetes (NIDDM)? *Am J Clin Nutr* 1986;43:16.

21. Coulston AM, Hollenbeck CB, Donner CC, et al: Metabolic effects of added dietary sucrose on individuals with non-insulin-dependent diabetes mellitus (NIDDM). *Metabolism* 1985;34:962-966.

22. Bantle JP, Laine DC, Thomas JW: Metabolic effects of dietary fructose and sucrose in Type I and II diabetic subjects. *JAMA* 1986;256:3241-3246.

23. Peterson DB, Lambert J, Gerrig S, et al: Sucrose in the diet of diabetic patients—just another carbohydrate? *Diabetologia* 1986;29:216-220.

24. National Academy of Sciences/National Research Council: *Recommended Dietary Allowances*, ed 9. Washington, DC, National Academy of Sciences/National Research Council, 1980.

25. Position of the American Dietetic Association: Appropriate use of nutritive and non-nutritive sweeteners: Position and summary. *J Am Diet Assoc* 1987;87:1689-1694.

26. American Diabetes Association: Position statement: Use of noncaloric sweeteners. *Diabetes Care* 1987;10:546.

27. Franz M, Krosnick A, Maschak-Carey BJ, et al: *Goals for Diabetes Education*. Alexandria, Va, American Diabetes Association, 1986.

28. Diabetes Care and Education Practice Group: The American Dietetic Association: *Meal Planning Approaches in the Nutrition Management of the Person with Diabetes*. Chicago, American Dietetic Association, 1987.

29. Franz M, Barr P, Holler H, et al: Exchange lists: Revised 1986. *J Am Diet Assoc* 1987;87:28-36.

30. International Diabetes Center: *Low Literacy Series*. Minneapolis, Diabetes Center, Inc, 1988.

31. American Diabetes Association: *Eating Health Foods*. Alexandria, Va, American Diabetes Association, 1988.

32. American Diabetes Association and The American Dietetic Association: *A Guide for Professionals: Diabetes Nutrition and Meal Planning*. Alexandria, Va, American Diabetes Association, 1988, pp 29,30.

33. Franz MJ, Hedding BK, Holtmeier K, et al: *A Guide to Healthy Eating*. Minneapolis, Diabetes Center, Inc, 1985, pp 8-10,54.

34. Monk A, Adolphson S, Hollander P, et al: *Managing Type II Diabetes: Your Invitation to a Healthier Lifestyle*. Minneapolis, Diabetes Center, Inc, 1988, pp 36,37,46-48,50.

35. Franz MJ: *Adding Fiber to Your Diet*. Minneapolis, Diabetes Center, Inc, 1988, pp 4,5.

36. Monk A, Franz MJ: *Convenience Food Facts*. Minneapolis, Diabetes Center, Inc, 1987, pp VI-XIV.

# The Role of Exercise in Type II Diabetes

Stephen H. Schneider, MD
Associate Professor of Medicine
Division of Endocrinology, Metabolism,
and Nutrition
UMDNJ—Robert Wood Johnson Medical School
New Brunswick, NJ

The preferred initial approach to treating Type II diabetes mellitus has traditionally consisted of diet and the casual encouragement of exercise. Although weight reduction is clearly the most effective therapy for the obese patient, the potential of exercise for improving insulin sensitivity and accelerate weight loss suggests that regular exercise should be a useful adjunct to dietary therapy (see Table 1).[1-5]

Table 1

| Potential Benefits of Exercise in Type I Diabetes |
|---|
| Enhanced insulin sensitivity |
| Decreased plasma-insulin levels |
| Improved glycemic control |
| Decreased levels of VLDL and LDL |
| Improved coagulation profile |
| Reduced blood pressure |
| More efficient myocardial work |
| Weight reduction |
| Enhanced self-image |

## Effects on Glycemic Control

Regular exercise enhances the disposal of a glucose load by at least two distinct mechanisms. Exercise at high intensity or exercise against resistance results in an improved ability to dispose of a glucose load in direct proportion to the associated increase of muscle mass.[6] Glucose disposal per kilogram of muscle is not enhanced. While this effect can be quite substantial for some patients, few patients with Type II diabetes are willing or able to exercise regularly at the necessary intensity. Nevertheless, aerobic exercise programs of more modest intensity have been shown to improve glucose tolerance and insulin sensitivity with little, if any, change in body composition. In general, improvements in glycosylated hemoglobin equaling 10 percent to 20 percent of baseline values are typical. Improved glucose control is related primarily to the residual effects of individual exercise sessions. The improved glucose disposal and increased sensitivity to insulin that follow a single bout of moderately intense exercise can last from 12 to 72 hours.[7-9] Therefore, exercising every other day usually results in a sustained improvement of blood glucose levels. The aerobically trained state per se seems to confer little additional benefit. Following many months of physical training, glucose disposal returns to the pretrained state within 72 hours after the last bout of exercise, and a single moderately intense exercise session will completely restore glucose disposal to the level achieved during training.

The mechanisms responsible for improved insulin sensitivity following exercise are poorly understood. Changes in the number and affinity of insulin receptors have been suggested, but attention is now focused on such postreceptor mechanisms as the increase in the number of glucose-transport units following physical activity.[10] While dietary therapy and weight reduction appear to improve glucose disposal, primarily by enhancing glucose oxidation, exercise enhances glucose disposal by increasing its storage in skeletal muscle. This difference in the fate of glucose suggests that the combination of diet and exercise might be particularly beneficial in enhancing glycemic control in the obese diabetic.[11]

### Prevention of Atherosclerotic Cardiovascular Disease

Patients with Type II diabetes suffer from accelerated atherosclerosis with an increased incidence of premature coronary artery disease, stroke, and peripheral vascular disease. Epidemiologic evidence and animal studies suggest that regular exercise offers protection against atherosclerosis in the general population.[12] No direct evidence is available for diabetic patients. Nevertheless, many known risk factors occur more commonly in Type II diabetes and may be favorably influenced by regular exercise.

### Lipids

Studies of Type II patients as well as normal individuals confirm significant hypolipidemic effects of regular exercise. These most often include a reduction of VLDL and LDL. Changes in LDL and HDL cholesterol are generally unimpressive. Hypolipidemic effects are at least partly independent of weight reduction and, as is true with glucose disposal, may dissipate rapidly when regular exercise is discontinued.[13,14] It appears that exercise of higher intensity or duration than most patients are willing or able to perform, may be necessary to produce effects on HDL cholesterol levels.

### Coagulation System

Coagulation abnormalities that are common in Type II diabetes include increased fibrinogen, factor VIII complex, platelet turnover, increased thromboxane/prostacycline ratio, and impaired fibrinolysis. Recent studies of the effects of regular exercise on these abnormalities are limited but suggest modest improvement in some parameters, particularly fibrinolytic activity.[15]

### Hyperinsulinemia

Epidemiologic studies have confirmed a close association between chronic hyperinsulinemia and accelerated atherosclerosis. This is particularly worrisome for the Type II patient, who often has endogenous hyperinsulinemia as a result of significant insulin resistance. Regular exercise has been shown to be effective in improving insulin sensitivity, producing the same or better glucose control with significantly lower ambient insulin levels.

### Hemodynamics

Patients with diabetes suffer from an increased incidence of hypertension and, often, subtle myocardial dysfunction. The hemodynamic benefits of regular exercise, including modest decreases in blood pressure[16] and more efficient myocardial work, have been shown to occur in patients with Type II diabetes as well as in normal individuals.

### Psychological Benefits

Regular exercise can result in an improved self-image and might be expected to enhance compliance with dietary regimens.

### Complications of Exercise

While exercise can be an effective therapeutic tool for Type II patients, there are associated risks (see Table 2).

### General Risks

Motor and sensory neuropathy and peripheral

Table 2

**Complications of Exercise**

**General**
Foot injuries and ulceration
Degenerative joint disease

**Cardiovascular**
Silent ischemia
Myocardial infarction
Hypertension
Orthostatic hypotension
Proteinuria
Retinal hemorrhage
Acceleration of microvascular lesions

**Metabolic**
Worsening metabolic control
Hypoglycemia

vascular disease may place the diabetic patient at high risk for foot injury or ulceration. Careful attention must be paid to selecting appropriate footwear, and patients must be taught to inspect their feet after exercising. Loss of proprioception may cause abnormal joint motion with an acceleration of degenerative joint disease. For some patients, exercises such as jogging are associated with significant foot trauma and may be contraindicated.

### Cardiovascular

The high incidence of silent ischemia in patients with Type II diabetes makes careful assessment of the cardiovascular system mandatory before initiating an exercise program.[17] In addition, postexercise orthostatic hypotension may be found in sedentary diabetics, even in the absence of clinically evident autonomic neuropathy. This tends to improve with physical training. Some patients have an exaggerated hypertensive response to submaximal exercise. High-intensity exercises that are associated with large elevations in systolic blood pressure or that involve Valsalva's maneuvers, are a major cause of retinal hemorrhage in patients with proliferative retinopathy. In addition, most patients who have long-standing diabetes develop transient proteinuria following high-intensity exercise. This is related to the degree of systolic hypertension that occurs during the activity.[18] Whether the hemodynamic and hormonal changes induced by intense exercise result in an acceleration of underlying nephropathy and/or retinopathy is currently unclear.

### Metabolic Risks

Metabolic complications are rare in patients with Type II diabetes mellitus but they may occur. Insulin-deficient patients whose fasting glucose levels are greater than 250 mg/dL may develop more severe hyperglycemia during exercise.[19] This is probably the result of a blunted increase in glucose uptake by the exercising muscle as well as an overproduction of glucose by the liver. Such individuals should be warned not to engage in exercise until they are better insulinized.

During or after exercise, hypoglycemia is relatively uncommon in Type II diabetes but may occur in patients on insulin or oral hypoglycemic agents.[20] Accelerated absorption of insulin from the injection site can occur when the underlying muscle is exercised. This is of importance only when exercise closely follows the injection of short-acting insulin.

Normally, plasma insulin levels fall during exercise and levels of catecholamines and glucagon increase. This restrains peripheral glucose utilization and enhances hepatic glucose production. Patients who take insulin and many patients who use oral agents are unable to decrease their plasma insulin levels appropriately. In addition, some patients may have an impaired counterregulatory response. Commonly used drugs, including $\beta$-adrenergic blocking agents, may exacerbate hypoglycemia during exercise in this subset of individuals.[21]

Exercise-related hypoglycemia often can be prevented by decreasing the dose of regular insulin that precedes exercise. The necessary reduction is variable and may range between 50 percent and 100 percent. Alternatively, consuming an additional 15 g to 30 g of carbohydrates for every 30 minutes of brisk physical activity may be useful, and is easier for most patients than alteration of insulin regimens. Because gastric hypertonicity causes gastroparesis, which impairs exercise capacity, no more than 30 g of carbohydrate should be consumed during every 30 minutes of exercise. Patients should be taught that hypoglycemia may occur many hours following exercise.[22] Those who are prone to these delayed reactions should consume an additional snack, such as low-fat milk, after exercise, and should probably avoid exercise in the evening. It is good practice for all diabetic patients to carry clear identification along with some form of rapidly absorbable carbohydrate when exercising away from home.

## FORMULATING AN EXERCISE REGIMEN

### How to Select Patients for Exercise Programs

Active young patients who have no complications and whose diabetes is of short duration require only basic education to continue their usual activities. Unfortunately, most Type II patients are relatively sedentary and will require a monitored program and formal preexercise evaluation. Such programs require considerable effort on the part of patients, physicians, nurses, and exercise physiologists. As a result, consideration should be given to identifying subgroups of patients who are most likely to benefit.

In general, patients with mild Type II diabetes, indicated by fasting glucose levels below 200 mg/dL to 220 mg/dL, and patients with greater degrees of hyperinsulinemia and insulin resistance, show the most substantial metabolic improvement. In our own experience, long-term compliance is often poor among patients with high levels of anxiety at the time exercise is initiated. As a result, we generally delay entry into our program for at least a month following diagnosis. For patients with strong type-A personality traits, particularly if the motivation to enter the program comes only from the physician, compliance is especially poor. Contrary to common belief, female and elderly patients do extremely well in monitored exercise programs. They usually have high compliance rates and excellent metabolic responses.

### Preexercise Evaluations

Physically active younger patients with a short duration of diabetes rarely require formal exercise evaluations. On the other hand, previously sedentary patients over 35 years of age, and younger patients with multiple cardiovascular risk factors (or duration of diabetes greater than 10 years) warrant some form of exercise testing before engaging in a serious training regimen (see Table 3). In addition, patients with established vascular or neuropathic complications of diabetes may require further assessments. Screening prior to establishing exercise recommendations should include the following:

1. Thorough history and physical examination to identify microvascular, macrovascular, and neuropathic complications of diabetes along with some assessment of body composition.

Table 3

**Evaluations Prior to Formulation of Exercise Regimens**

- History and physical exam
- Review of medications (ie, β-adrenergic blockers)
- Cardiovascular risk-factor profile
- Fundoscopic examination
- Podiatric evaluation
- Assessment of:
    - Glucose control and adequacy of insulinization
    - Sensory-motor neuropathy
    - Autonomic neuropathy
- Estimate of body composition
- Formal exercise testing:
    - Pulse and blood-pressure response
    - EKG monitoring
    - Postexercise orthostatic blood pressure
    - Hypoglycemic response to exercise
    - Proteinuria following exercise (qualitative)

2. An assessment of cardiovascular risk factors, including smoking, hypercholesterolemia, and hypertension.

3. An assessment of autonomic nervous system function. This should include symptoms of delayed gastric emptying, urinary bladder dysfunction, impotence, and determinations of blood pressure while lying and standing. In addition, noninvasive objective tests of cardiac autonomic reflexes can be performed in the office. Two tests we have found useful include determination of RR-interval variation during respiratory cycles and heart rate during Valsalva's maneuver.

### RR-Interval Variation

During EKG monitoring the patient is asked to take full five-second inspirations and five-second expirations with no breath holding in between, for five consecutive respiratory cycles. A mean heart-rate variation less than 10 beats per minute (BPM) during the respiratory cycle is considered abnormal; 10 to 15 BPM is indeterminate, and greater than 15 BPM is within the normal range.[23]

### Valsalva's Maneuver

Valsalva's maneuver can be performed by having the subject blow out against a measured resistance of 40 mm Hg for 15 seconds. Heart rate is continuously recorded by EKG and a ratio of the maximum heart rate during attempted expiration and the minimum heart rate following the maneuver are obtained. The lower limit of normal for the ratio between the longest RR interval after Valsalva's maneuver to the shortest RR interval during Valsalva's maneuver is 1.5. This test should probably be avoided for patients with proliferative retinopathy.[24]

### Exercise Testing

Subjects with no obvious clinical contraindications should undergo some form of submaximal or, preferably, maximal exercise testing. In addition to identifying patients with silent ischemia, testing may reveal individuals who have an exaggerated hypertensive response to exercise or who are prone to developing postexercise orthostatic hypertension.

Testing should be done following at least five minutes of warm-up activity. Subsequently, work loads can be increased at five-minute intervals until the patient feels exhausted or a predetermined level of work is obtained. At a minimum, EKG monitoring as well as pulse and blood pressure determinations should be performed at five-minute intervals. Additional information that may be of value for some patients includes oxygen uptake and carbon dioxide production as well as ratings of perceived exertion.[25]

Ideally, patients should be exercised to an intensity approximating their maximal work capacity. As a practical matter, this will be difficult or dangerous for many patients. For clinical purposes, it is often necessary to estimate maximal exercise capacity, using any of a number of simple nomograms (see Table 4 and Figure 1).[26-28] Even more simply, an approximation of maximal heart rate can be obtained by subtracting a patient's age from 220. However, this is an inaccurate means of assessing exercise capacity, and tables derived from normal populations may overestimate maximal pulse and oxygen consumption in diabetic patients by 10 percent to 15 percent, particularly those with even mild autonomic neuropathy. The exercise regimen for patients with diabetes is similar in most respects to recommendations for the general public. Appendix A demonstrates how to use exercise testing results to create a program for a sample patient.

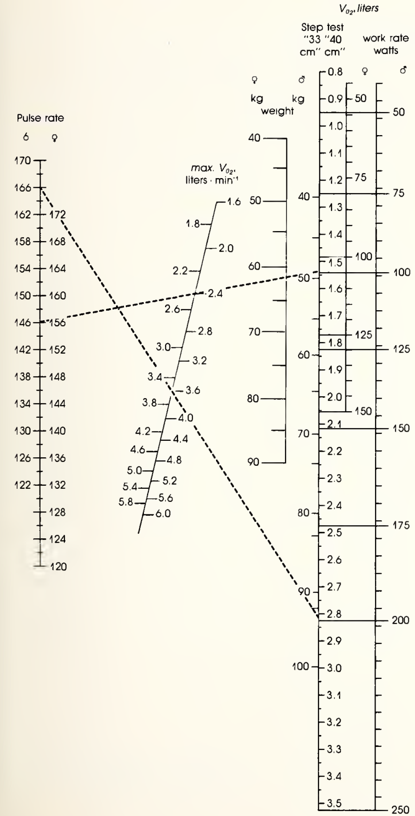
### Types of Exercise

In general, aerobic exercise of moderate intensity is recommended (see Appendix B). This consists of activities that call for repetitive submaximal contractions of large muscle groups. Classic examples include swimming, cycling, rowing, skiing, jogging, and walking. The activity selected needs to be highly individualized. No program will succeed that utilizes regimens the patient does not find enjoyable or in which the patient cannot become reasonably competent. On the other hand, characteristics of individual patients may determine preferred types of exercise. Obese patients often find swimming to be an activity they can readily perform. Yet, such individuals often find jogging to

This adjusted nomogram can be used to calculate maximal oxygen uptake from submaximal pulse rate and $O_2$-uptake values (cycling, running or walking, and step test). In tests without direct $O_2$-uptake measurement, it can be estimated by reading horizontally from the "body weight" scale (step test) or "work rate" scale (cycle test) to the "$O_2$-uptake" scale. The point on the $O_2$-uptake scale ($VO_2$, liters) shall be connected with the corresponding point on the pulse rate scale, and the predicted maximal $O_2$ uptake read on the middle scale. A female subject (61 kg) reaches a heart rate of 156 at step test; predicted max. $VO_2 = 2.4$ liters $\cdot \text{min}^{-1}$. A male subject reaches a heart rate of 166 at cycling test on a work rate of 200 Watts; predicted max. $VO_2 = 3.6$ liters $\cdot \text{min}^{-1}$ (exemplified by dotted lines). *[From Astrand]*

Figure 1

## Nomogram for Estimating Maximal Exercise Capacity



### Correction Factor for use with Figure 1 and Table 4

| Age | Factor | Max heart rate | Factor |
|---|---|---|---|
| 15 | 1.10 | 210 | 1.12 |
| 25 | 1.00 | 200 | 1.00 |
| 35 | 0.87 | 190 | 0.93 |
| 40 | 0.83 | 180 | 0.83 |
| 45 | 0.78 | 170 | 0.75 |
| 50 | 0.75 | 160 | 0.69 |
| 55 | 0.71 | 150 | 0.64 |
| 60 | 0.68 | | |
| 65 | 0.65 | | |

These factors should be used for correcting predicted maximal oxygen uptake (1) when the subject is over 30 to 35 years of age or (2) when the subject's maximal heart rate is known. The actual factor should be multiplied by the value that is obtained, from both this nomogram and Table 4.

Note: More sophisticated correction can be performed using equation found in Reference 18.

## Tabular Nomograms for Estimating Maximal Exercise Capacity

**(use with correction factor from Figure 1)**

Table 4

**Women**

| | (Maximal oxygen uptake, liters/min) | | | | | | (Maximal oxygen uptake, liters/min) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Heart rate | 300 kpm/min 50W | 450 kpm/min 75W | 600 kpm/min 100W | 750 kpm/min 125W | 900 kpm/min 150W | Heart rate | 300 kpm/min 50W | 450 kpm/min 75W | 600 kpm/min 100W | 750 kpm/min 125W | 900 kpm/min 150W |
| 120 | 2.6 | 3.4 | 4.1 | 4.8 | | 148 | 1.6 | 2.1 | 2.6 | 3.1 | 3.6 |
| 121 | 2.5 | 3.3 | 4.0 | 4.8 | | 149 | | 2.1 | 2.6 | 3.0 | 3.5 |
| 122 | 2.5 | 3.2 | 3.9 | 4.7 | | 150 | | 2.0 | 2.5 | 3.0 | 3.5 |
| 123 | 2.4 | 3.1 | 3.9 | 4.6 | | 151 | | 2.0 | 2.5 | 3.0 | 3.4 |
| 124 | 2.4 | 3.1 | 3.8 | 4.5 | | 152 | | 2.0 | 2.5 | 2.9 | 3.4 |
| 125 | 2.3 | 3.0 | 3.7 | 4.4 | | 153 | | 2.0 | 2.4 | 2.9 | 3.3 |
| 126 | 2.3 | 3.0 | 3.6 | 4.3 | | 154 | | 2.0 | 2.4 | 2.8 | 3.3 |
| 127 | 2.2 | 2.9 | 3.5 | 4.2 | | 155 | | 1.9 | 2.4 | 2.8 | 3.2 |
| 128 | 2.2 | 2.8 | 3.5 | 4.2 | 4.8 | 156 | | 1.9 | 2.3 | 2.8 | 3.2 |
| 129 | 2.2 | 2.8 | 3.4 | 4.1 | 4.8 | 157 | | 1.9 | 2.3 | 2.7 | 3.2 |
| 130 | 2.1 | 2.7 | 3.4 | 4.0 | 4.7 | 158 | | 1.8 | 2.3 | 2.7 | 3.1 |
| 131 | 2.1 | 2.7 | 3.4 | 4.0 | 4.6 | 159 | | 1.8 | 2.2 | 2.7 | 3.1 |
| 132 | 2.0 | 2.7 | 3.3 | 3.9 | 4.5 | 160 | | 1.8 | 2.2 | 2.6 | 3.0 |
| 133 | 2.0 | 2.6 | 3.2 | 3.8 | 4.4 | 161 | | 1.8 | 2.2 | 2.6 | 3.0 |
| 134 | 2.0 | 2.6 | 3.2 | 3.8 | 4.4 | 162 | | 1.8 | 2.2 | 2.6 | 3.0 |
| 135 | 2.0 | 2.6 | 3.1 | 3.7 | 4.3 | 163 | | 1.7 | 2.2 | 2.6 | 2.9 |
| 136 | 1.9 | 2.5 | 3.1 | 3.6 | 4.2 | 164 | | 1.7 | 2.1 | 2.5 | 2.9 |
| 137 | 1.9 | 2.5 | 3.0 | 3.6 | 4.2 | 165 | | 1.7 | 2.1 | 2.5 | 2.9 |
| 138 | 1.8 | 2.4 | 3.0 | 3.5 | 4.1 | 166 | | 1.7 | 2.1 | 2.5 | 2.8 |
| 139 | 1.8 | 2.4 | 2.9 | 3.5 | 4.0 | 167 | | 1.6 | 2.1 | 2.4 | 2.8 |
| 140 | 1.8 | 2.4 | 2.8 | 3.4 | 4.0 | 168 | | 1.6 | 2.0 | 2.4 | 2.8 |
| 141 | 1.8 | 2.3 | 2.8 | 3.4 | 3.9 | 169 | | 1.6 | 2.0 | 2.4 | 2.8 |
| 142 | 1.7 | 2.3 | 2.8 | 3.3 | 3.9 | 170 | | 1.6 | 2.0 | 2.4 | 2.7 |
| 143 | 1.7 | 2.2 | 2.7 | 3.3 | 3.8 | | | | | | |
| 144 | 1.7 | 2.2 | 2.7 | 3.2 | 3.8 | | | | | | |
| 145 | 1.6 | 2.2 | 2.7 | 3.2 | 3.7 | | | | | | |
| 146 | 1.6 | 2.2 | 2.6 | 3.2 | 3.7 | | | | | | |
| 147 | 1.6 | 2.1 | 2.6 | 3.1 | 3.6 | | | | | | |

W = watts

From a nomogram by I Astrand (1960)

**Men**

| Heart rate | (Maximal oxygen uptake, liters/min) | | | | |
|---|---|---|---|---|---|
| | 300 kpm/min 50W | 600 kpm/min 100W | 900 kpm/min 150W | 1200 kpm/min 200W | 1500 kpm/min 250W |
| 120 | 2.2 | 3.5 | 4.8 | | |
| 121 | 2.2 | 3.4 | 4.7 | | |
| 122 | 2.2 | 3.4 | 4.6 | | |
| 123 | 2.1 | 3.4 | 4.6 | | |
| 124 | 2.1 | 3.3 | 4.5 | 6.0 | |
| 125 | 2.0 | 3.2 | 4.4 | 5.9 | |
| 126 | 2.0 | 3.2 | 4.4 | 5.8 | |
| 127 | 2.0 | 3.1 | 4.3 | 5.7 | |
| 128 | 2.0 | 3.1 | 4.2 | 5.6 | |
| 129 | 1.9 | 3.0 | 4.2 | 5.6 | |
| 130 | 1.9 | 3.0 | 4.1 | 5.5 | |
| 131 | 1.9 | 2.9 | 4.0 | 5.4 | |
| 132 | 1.8 | 2.9 | 4.0 | 5.3 | |
| 133 | 1.8 | 2.8 | 3.9 | 5.3 | |
| 134 | 1.8 | 2.8 | 3.9 | 5.2 | |
| 135 | 1.7 | 2.8 | 3.8 | 5.1 | |
| 136 | 1.7 | 2.7 | 3.8 | 5.0 | |
| 137 | 1.7 | 2.7 | 3.7 | 5.0 | |
| 138 | 1.6 | 2.7 | 3.7 | 4.9 | |
| 139 | 1.6 | 2.6 | 3.6 | 4.8 | |
| 140 | 1.6 | 2.6 | 3.6 | 4.8 | 6.0 |
| 141 | | 2.6 | 3.5 | 4.7 | 5.9 |
| 142 | | 2.5 | 3.5 | 4.6 | 5.8 |
| 143 | | 2.5 | 3.4 | 4.6 | 5.7 |
| 144 | | 2.5 | 3.4 | 4.5 | 5.7 |
| 145 | | 2.4 | 3.4 | 4.5 | 5.6 |
| 146 | | 2.4 | 3.3 | 4.4 | 5.6 |
| 147 | | 2.4 | 3.3 | 4.4 | 5.5 |
| 148 | | 2.4 | 3.2 | 4.3 | 5.4 |
| 149 | | 2.3 | 3.2 | 4.3 | 5.4 |
| 150 | | 2.3 | 3.2 | 4.2 | 5.3 |
| 151 | | 2.3 | 3.1 | 4.2 | 5.2 |
| 152 | | 2.3 | 3.1 | 4.1 | 5.2 |
| 153 | | 2.2 | 3.0 | 4.1 | 5.1 |
| 154 | | 2.2 | 3.0 | 4.0 | 5.1 |
| 155 | | 2.2 | 3.0 | 4.0 | 5.0 |
| 156 | | 2.2 | 2.9 | 4.0 | 5.0 |
| 157 | | 2.1 | 2.9 | 3.9 | 4.9 |
| 158 | | 2.1 | 2.9 | 3.9 | 4.9 |
| 159 | | 2.1 | 2.8 | 3.8 | 4.8 |
| 160 | | 2.1 | 2.8 | 3.8 | 4.8 |
| 161 | | 2.0 | 2.8 | 3.7 | 4.7 |
| 162 | | 2.0 | 2.8 | 3.7 | 4.6 |
| 163 | | 2.0 | 2.8 | 3.7 | 4.6 |
| 164 | | 2.0 | 2.7 | 3.6 | 4.5 |
| 165 | | 2.0 | 2.7 | 3.6 | 4.5 |
| 166 | | 1.9 | 2.7 | 3.6 | 4.5 |
| 167 | | 1.9 | 2.6 | 3.5 | 4.4 |
| 168 | | 1.9 | 2.6 | 3.5 | 4.4 |
| 169 | | 1.9 | 2.6 | 3.5 | 4.3 |
| 170 | | 1.8 | 2.6 | 3.4 | 4.3 |

W = watts

From a nomogram by I Astrand (1960)

be a difficult activity. Elderly patients often prefer walking programs as a form of exercise. Stationary bicycling is another excellent exercise but it has a relatively low long-term compliance rate.

Certain complications may place limitations on activities patients can pursue. For example, patients with proliferative retinopathy should avoid such exercises as racquetball or tennis that can result in eye injury, unless protective goggles are worn to ensure safety during these activities.

Patients with established microvascular disease should probably avoid scuba diving and other activities that expose them to changes in atmospheric pressure. They should also avoid intense resistance exercises, including weight lifting, which may result in retinal hemorrhage and proteinuria. Resistance exercises need not be discouraged for younger patients with no established vascular complications. However, because of the high rate of musculoskeletal complications, the potential adverse effects associated with wide swings of blood pressure, and the small likelihood that this type of activity will be continued into later life, aerobic activity is preferred. (see Table 5).

Table 5

**Exercise Recommendations**

I) **Type**
Aerobic preferred (ie, swimming, cycling, walking)

II) **Warm-up**
Stretching and light exercise ≃ 10 minutes (avoid Valsalva's maneuver)

III) **Intensity**
Start at about 50% maximum; work up to 60% to 75% over 4 to 6 weeks

IV) **Duration**
20 to 40 minutes
60 to 90 seconds (rest periods permissible)

V) **Frequency**
> 3x per week for glycemic control
> 5x per week for weight reduction

VI) **Warm-down**
≃ 10 minutes at 20% to 30% intensity of exercise session

### Timing of Exercise Sessions

Exercise in the morning will be most effective in enhancing carbohydrate disposal in subsequent meals throughout the day. This is because the effects of exertion on glucose disposal are maximal for the first 12 hours following activity. In addition, evidence suggests that because the hypoglycemic effect of exercise is less abrupt in the morning, the risk of postexercise hypoglycemia is reduced. Exercise late in the evening is probably ill advised for insulin-treated patients as it enhances the risk of early-morning hypoglycemic episodes. There is some evidence that exercise more efficiently enhances glucose disposal when it is performed immediately following a meal. This advantage needs to be weighed against the significantly greater cardiovascular stress of postprandial physical activity.

### Warm-up

All individuals should warm up prior to every session. The warm-up period can be divided into two parts. In the first, all individuals should undergo at least five minutes of stretching activities (Appendix C).[29] This can substantially reduce the incidence of musculoskeletal injuries. A common mistake in stretching involves using short, jerky motions that actually increase muscle tightness. Breath holding during stretching can result in Valsalva-type activity and should be avoided. After stretching, an individual should engage in five minutes of a mild aerobic warm-up.

### Intensity

The intensity of exercise depends on an individual's cardiovascular fitness and hemodynamic response. In general, exercise is prescribed according to a target pulse rate based on basal and maximal pulse rates. The basal pulse is measured by the patient before rising from bed on a number of mornings. For clinical purposes, it is often necessary to estimate maximal exercise capacity from a submaximal response. The metabolic response to physical exertion appears to be determined primarily by relative, not absolute, aerobic work intensity. Thus, the responses that occur with a sedentary elderly patient during a brisk walk can be similar to those produced by a fit younger person during a brisk run, that is if each represents roughly the same percent of the individual's aerobic capacity.

In general, exercise programs are initiated at 50 percent to 60 percent of an individual's maximal aerobic capacity. This can be estimated by taking half the difference between maximal and basal heart rate and adding the result to the basal heart rate (see Appendix A). Exercise intensity can then be gradually increased over a four- to six-week period to between 70 percent and 75 percent of an individual's capacity. Recommended intensity would be decreased by such complications as silent ischemia, microvascular disease, or an exaggerated hypertensive response. While there are no good prospective studies to support these guidelines, it appears prudent to avoid exercises that raise systolic blood pressure above 180 mm Hg to 200 mm Hg for prolonged periods of time.

Patients can easily be taught to monitor their own pulse rates. For rare individuals who cannot do so because of severe sensory neuropathy, a number of inexpensive devices are available to help. For some forms of exercise, it may be necessary to take the pulse following exertion. The pulse can be taken immediately after exercise for 10 seconds and then multiplied by six to arrive at a reasonable approximation of the pulse rate during the exercise. Individuals should allow at least five minutes before reassessing pulse rate when intensity of exercise is changed. While the heart rate can be a very practical means of estimating appropriate exercise intensity, in some situations it may be misleading. These situations include exercise in extremes of heat or cold, exercise during the absorptive phase following a large meal or after alcohol consumption, and exercise in the presence of such drugs as $\beta$-adrenergic blockers. For patients with severe microvascular disease, for whom the blood pressure response is particularly important, it may be worthwhile to measure the response to a recommended exercise regimen on more than one occasion. Such patients should probably not take part in strenuous exercise without expert supervision.

### Warm-down

Many serious cardiac arrhythmias appear to occur in the first five to 10 minutes after exercise. A five- to 10-minute warm-down period at 20 percent to 30 percent of exercise exertion may not only decrease the risk of cardiac arrhythmias, but will also accelerate the resolution of muscle fatigue.

### Duration

The duration of exercise should be at least 20 minutes. In our experience, exercise that lasts longer than 40 to 45 minutes is associated with an unacceptable incidence of musculoskeletal complaints among older patients. Studies have shown that brief rest periods (60 to 90 seconds) at five-minute intervals result in little impairment of beneficial effects.

### Frequency

Because much of the metabolic benefit of regular exercise results from the residual effects of each exercise bout, the more frequent the exercise the better the metabolic response. In our experience, exercising less than three times a week is ineffective as a therapeutic tool regardless of intensity. Higher intensity cannot compensate for decreased frequency. Exercising three or four times a week is necessary for significant metabolic benefits, and if weight reduction is important, exercising at least five times per week may be necessary. On the other hand, in older sedentary populations, exercising more than four times per week may be associated with a relatively high incidence of musculoskeletal complaints.

### Maintenance Phase

Patients will soon discover that in order to maintain the initial target heart rate, they will have to exercise at gradually increasing work loads. Because the maximal heart rate changes little, if at all, with conditioning, the target pulse will remain the same for long periods of time.

The training effects of regular exercise on metabolic parameters appear to be well established in the first six weeks of physical training. While some additional benefit may occur over the subsequent three to six months, it is generally modest and may be related to changes in body composition.

Long-term compliance with physical exercise programs is subject to many of the same difficulties associated with other attempts at maintaining weight reduction. Helpful strategies include participation of the spouse, and group sessions that encourage social interaction. Selection of activities in which a patient can develop a reasonable degree of proficiency and enjoyment are essential.

### Patient Feedback

As with other attempts at behavior modification, meaningful feedback from patients is required for maintaining compliance. This also facilitates appropriate modification of the exercise program. Reasonable short- and long-term goals should be established prior to initiating the program. "Ideal" fitness levels can be found in Table 6.[30] Feedback for a combined exercise and dietary program should comprise at least three components:

Metabolic studies—These should include indices of glycemic control, including glycosylated hemoglobin, as well as such measures as serum triglyceride levels, which are likely to respond to exercise.

Weight and body composition—Patients who engage in serious exercise programs and other attempts at weight loss need to be taught how to interpret their progress. Changes in body composition that correlate with metabolic and hemodynamic improvements may occur in the face of little or no reduction in total body weight. In addition, body fat may be redistributed from the abdominal area towards the arms, legs, and thighs. In and of itself, this may be beneficial.[31] Where possible, some index of body composition should be measured at regular intervals. Caliper measurements are often used for this purpose, but are rather insensitive. Newer equipment now becoming available may make routine assessment of body composition more reasonable for the practicing physician.

Physical fitness assessment—It is generally impractical to do standard exercise testing at regular intervals. As a result, more simple indices of fitness are generally used. A decrease in resting pulse rate is often used as an index of physical fitness. However, in diabetes, early subclinical parasympathetic neuropathy results in a relatively rapid resting heart rate, which fails to decrease in response to training. Nevertheless, the pulse rate and systolic blood pressure for any given submaximal absolute workload will decrease progressively as training occurs. A quick five-minute exercise session at a relatively low constant absolute workload, can be performed at regular intervals in the office to monitor these changes. We generally evaluate patients after six and 12 weeks of training and every three months thereafter.

### The Use of Exercise as an Adjunct to Diet

One reason physicians recommend exercise for patients with Type II diabetes is their belief that regular exercise will aid in achieving a greater degree of weight reduction. Exercise might be effective in this respect because:

- It encourages direct utilization of calories
- It produces a sustained increase in metabolic rate that may persist for some time after the exertion
- It can be responsible for positive behavioral and psychological changes, including improved body image and awareness

Unfortunately, as a tool for weight reduction, physical training has generally been disappointing. There are a number of possible explanations for this apparent lack of efficacy. First, the number of calories directly consumed during moderate physical activity is actually quite modest, rarely exceeding 300 calories per session (see Appendix B).[32] In addition, moderate exercise for mildly overweight individuals may increase appetite and caloric intake. This increased intake partially counteracts the negative caloric balance during the exertion. Psychological factors may also play a role in this process because patients often reward themselves for physical activity with additional food. Finally, increased caloric expenditure during and immedi-

Table 6

**Target Values of Maximal Oxygen Uptake for Male and Female Populations of Different Ages**

| Group | Males | Females |
|---|---|---|
| General population* by age | $VO_2$ max ($mL \cdot kg^{-1} \cdot min^{-1}$) | |
| 10-14 | 47-51 | 47-51 |
| 15-19 | 49-56 | 44-52 |
| 20-29 | 43-52 | 37-46 |
| 30-39 | 39-48 | 33-42 |
| 40-49 | 36-44 | 30-38 |
| 50-59 | 34-41 | 28-35 |
| 60< | 31-38 | 25-32 |

*Values are desirable, not population norms.

From Wilmore and Costill.[30]

ately after exercise may be counteracted when the metabolic rate falls below baseline during other periods of the day, especially for patients on very-low-calorie diets.[33]

Exercise has an important but limited role as an adjunct to dietary therapy for weight reduction. In order for exercise to be effective, it must be performed regularly at least five times per week in conjunction with caloric restriction and other behavior-modification techniques. The combination of regular exercise and dietary therapy may result in changes of body composition and fat distribution that are highly beneficial to the patient but may not be reflected in terms of body weight. As a result, better patient education and somewhat more sophisticated monitoring of body composition are likely to be helpful in maintaining compliance.

### Acknowledgements

This work was supported in part by a National Institutes of Health Grant, "Physical Training and Vascular Risk Factors in Diabetes" (No. AM 26894).

This work was also supported in part by an American Diabetes Association Grant, "The Role of Impaired Secretion Glucagon and Epinephrine in Exercise-Induced Hypoglycemia in Type I Diabetes Mellitus."

### References

1. Horton ES: Role of management of exercise in diabetes mellitus. *Diabetes Care* 1988;11:201-211.

2. Kanj H, Schneider SH, Ruderman NB, et al: Exercise and the diabetic, in Horton FS, Terjung RJ, (eds): *Exercise Nutrition and Energy Metabolism.* New York, Macmillan Publishing Co, 1988;228-241.

3. Krotkiewski M, Lonnroth P, Manrwoukas K, et al: Effects on physical training of insulin secretion and effectiveness and glucose metabolism in obesity and Type 2 (non-insulin dependent) diabetes mellitus. *Diabetes Mellitus* 1985;28:881-890.

4. Reitman JS, Vasquez B, Klimes I, et al: Improvements of glucose homeostasis after exercise training in non-insulin dependent diabetes. *Diabetes Care* 1984;7:434-441.

5. NIH Consensus Development Conference Statement: *Diet and Exercise in Non-Insulin Dependent Diabetes Mellitus.* NIH Consensus Development Conference, 1986, 6 (8).

6. Miller WJ, Sherman WM, Ivy JL. Effect of strength training on glucose intolerance and post-glucose insulin response. *Med Sci Sports Exer* 1984;16:539-543.

7. Devlin JT, Hirshman M, Horton FD, et al: Enhanced peripheral and splanchnic insulin sensitivity in NIDDM men after a single bout of exercise. *Diabetes* 1987;36:434-439.

8. Holloszy JO, Schultz J, Kusnierkiewicz J, et al: Effects of exercise on glucose tolerance and insulin resistance. *Acta Med Scand* 1987;711:55-65.

9. Schneider SH, Amorosa LF, Khachadurian AK, et al: Studies on the mechanism of improved glucose control during regular exercise in Type 2 (non-insulin dependent) diabetes. *Diabetologia* 1984;26:355-360.

10. Vinten J, Norgarrd-Petersen L, Sonne B, et al: Effect of physical training on glucose transporters. *Biochem Biophys Acta* 1985;841:223-227.

11. Bogardus C, Ravussin E, Robbins DC, et al: Effects of physical training and diet therapy in carbohydrate metabolism in patients with glucose intolerance and non-insulin dependent diabetes mellitus. *Diabetes* 1984;33:311-318.

12. Schneider SH, Vitug A, Ruderman NB: Atherosclerosis and physical activity. *Diabetes Metab Rev* 1986;1:513-553.

13. Ruderman NB, Ganda OP, Johansen K: The effects of physical training on glucose tolerance and plasma lipids in maturity onset diabetes. *Diabetes* 1979;28(suppl 1):89-92.

14. Holloszy JO, Skinner JS, Toro G, et al: Effects of

a six month program of endurance exercise on the serum lipids of middle-aged men. *Am J Cardiol* 1964;14:753-760.

15. Schneider SH, Kim HC, Khachadurian AK, et al: mpaired fibrinolytic responses to exercise in Type I diabetes: Effects of exercise and physical training. *Metabolism* 1988;37:924-929.

16. Krotkiewski M, Mandroukas K, Sjostrom L, et al: Effects of long term physical training on body fat, metabolism, and blood pressure in obesity. *Metabolism* 1979;28:650-658.

17. Nesto RW, Phillips RT, Kett KG, et al: Angina and exertional myocardial ischemia in diabetic and non-diabetic patients: Assessment by exercise thalium scintigraphy. *Ann Intern Med* 1988;108:170-175.

18. Mogensen CE, Christensen CK, Vittinghus E: The stages in diabetic renal diseases with emphasis on the stage of incipient diabetic nephropathy. *Diabetes* 1983;32(suppl 2):64-78.

19. Kemmer FW, Berger M: Therapy and better quality of life: The dichotomous role of exercise in diabetes mellitus. *Diabetes Metab Rev* 1986;2:53-68.

20. Kemmer FW, Tacken M, Berger M: Mechanism of exercise induced hypoglycemia during sulfonylurea treatment. *Diabetes* 1987;36:1178-1182.

21. DeFeo P, Bolli G, Perriello G, et al: The adrenergic contribution to glucose counter-regulation in Type I diabetes mellitus: Dependence on alpha-cell function in mediation through beta adrenergic receptors. *Diabetes* 1983;32:887-893.

22. MacDonald MJ: Post exercise late-onset hypoglycemia in insulin-dependent diabetic patients. *Diabetes Care* 1987;10:584-588.

23. Genovely H, Pfeifer MA: R-R variation: The autonomic test of choice in diabetes. *Diabetes Metab Rev* 1988;4:255-271.

24. Edwing DJ, Campbell IW, Burt AA, et al: Vascular reflexes in diabetic autonomic neuropathy. *Lancet* 1973;2:1354-1356.

25. Schneider SH, Ruderman NB, Young JC: Exercise studies in the diabetics, in Larner J, Pohl D, Clark W (eds): *Methods in Diabetes Research, Volume II: Clinical Methods*. New York, Wiley Press, 1986, pp 406-423.

26. Astrand PO, Rodahl K: Deobold B, Van Dalen (eds): *Textbook of Work Physiology.* New York, McGraw-Hill, 1986, p 365.

27. Wilmore JH, Costill DL: *Training for Sport and Activity.* Boston: Allyn and Bacon, 1987.

28. Siconolf SF, Cullinane EM, Carleton RA, et al: Assessing $VO_2$ max in epidemiologic studies: modification of the Astrand-rhyming test. *Med Sci Sports Exerc* 1982;14:335-338.

29. Anderson B: How to Stretch. Adapted from *Stretching*. Bolinas, Ca, Shelter Publications, 1980.

30. Wilmore JH, Costill DL: *Training for Sport and Activity,* ed 3. Boston: Allyn and Bacon, 1987.

31. Bjorntorp P: Abdominal obesity and the development of non-insulin dependent diabetes mellitus. *Diabetes Metab Rev* 1988;4:615-627.

32. Fox S: *Preventive Cardiology Program*. Washington, DC, Georgetown University Medical Center.

33. Phinney SD, LaGrange BM, O'Connell M, et al: Effects of aerobic exercise on energy expenditure and nitrogen balance during very low calorie dieting. *Metabolism* 1988;37:758-765.

# Appendix A
# Generation of an Exercise Regimen

**Exercise Test**

PATIENT: J.D. SEX: M AGE: 53
WEIGHT: 174 lb HEIGHT: 5′ 8″ Dx: Mild retinopathy

| Workload (Watts) | Pulse (beats per min) | BP (mm Hg) |
|---|---|---|
| Rest | 72 | 132/85 |
| 50 | 110 | 164/90 |
| 75 | 125 | 184/90 |
| 100 | 145 | 200/94 |
| 125 | 170 | 220/96 |
| 50 | 141 | 144/90 |
| Rest | 110 | 128/94 |

|  |  |  |
|---|---|---|
| Resting pulse | 60 (mean of 3 before rising in the morning) |  |
| Glucose | preexercise: 188 | postexercise: 178 |
| Urine protein | preexercise: neg | postexercise: 2+ |
| Postexercise BP | lying: 130/92 | standing: 132/90 |
| EKG abnormalities | none |  |

**Target Pulse Calculations**

Maximal pulse - basal pulse = 110
(170) (60)

0.5 x 110 = 55 0.7 x 110 = 77

50% maximal work (pulse estimate) = 115

70% maximal work (pulse estimate) = 137

**Copy This Page for Patient Information**

**Regimen**

NAME: DATE:

Sessions per week: 4
Duration of session: 45 minutes
Time of day: morning
Session breakdown:

| Purpose | Type of activity | Duration |
|---|---|---|
| Stretching | as directed | 5 minutes |
| Warm-up | brisk walk<br>target pulse 90 | 5 minutes |
| **Training session:** | | |
| Stationary bicycle | target pulse 115 | 15 minutes |
| Slow down if desired | | 90 seconds |
| Stationary bicycle | target pulse 115 | 15 minutes |
| Warm-down: | | |
| Stationary bicycle | target pulse 90 | 5-10 minutes |

ADJUSTMENTS:
Target pulse should be gradually increased to 135 BPM over 4 to 6 weeks.

DIETARY ADJUSTMENTS:
10 AM snack 15 g carbohydrates

MEDICATION ADJUSTMENT:
Decrease morning dose of regular insulin from 10 U to 5 U on exercise days.

LIMITATIONS:
Do not exceed pulse of 140 BPM*. If you experience chest discomfort of any kind, lightheadedness or unusual shortness of breath, stop exercising and contact your physician as soon as possible.

---

*Note: Presence of retinopathy and proteinuria following exercise argue against exercise of greater intensity.

**Copy This Page for Patient Information**

## Do's and Don'ts for Exercising with Diabetes

### DO

Wear footwear that fits properly.
Consume 15 to 30 g of carbohydrates every ½ hour of rigorous exercise.
Warm up for five to 10 minutes before exercise.
Cool down five to 10 minutes after exercise.
Try to inject insulin away from the most active muscles.
Exercise regularly, at least three times a week.
Tell your doctor about all symptoms you experience during exercise.
Keep some rapidly absorbable carbohydrate with you at all times.
Wear identification as a diabetic when you exercise.
Exercise with a friend.
Have fun!

### DON'T

Exercise when it is extremely hot or cold.
Exercise when your fasting blood sugar is greater than 250 mg/dL.
Exercise when you have a fever.
Exercise if your resting pulse is unusually high.
Exceed your target pulse rate.
Exercise shortly after an insulin injection.
Exercise immediately after a heavy meal.
Exercise on an empty stomach.
Drink alcohol prior to exercise.

**Copy This Page for Patient Information**

**Patient's Progress Sheet***

NAME:
STANDARD WORKLOAD: 75 watts

| Item | Goal | Entry | 6 wks | 12 wks |
|---|---|---|---|---|
| Weight | 155 | 174 | 171 | 168 |
| Percent body fat | 15% | 25% | 24% | 22% |
| Resting heart rate | 60-80 | 78 | 76 | 76 |
| Pulse 75 watts | 92-97 | 108 | 98 | 92 |
| Resting systolic BP | <140 | 164 | 160 | 158 |
| BP 75 watts | <190 | 224 | 188 | 192 |
| FBS | <140 | 198 | 190 | 185 |
| $HgbA_1$ | <8.6% | 12.9 | 11.2 | 10.6 |
| Triglycerides | <150 | 165 | 104 | 96 |
| Cholesterol | <180 | 193 | 186 | 218 |

*Bring this flowchart with you to each visit.

**Patient's Diary**

| DATE | ACTIVITY | DURATION | RESTING PULSE | PULSE ACHIEVED | RECOVERY PULSE | SYMPTOMS |
|---|---|---|---|---|---|---|
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |

**Copy This Page for Patient Information**

# Appendix B

**Samuel M. Fox III, MD**
**Preventive Cardiology Program**
**Georgetown University Medical Center**

| Activity or Exercise | METs[1] | KCAL per h[2] | Cardio-respiratory Endurance | Muscle Strength, Power & Endurance | Coordination, Balance & Grace | Flexibility & Agility | Stress Management/[3] Diversion | Social Opportunities | Injury & Hazard Problems[4] |
|---|---|---|---|---|---|---|---|---|---|
| Archery | 2-3 | 150-250 | + walking | +++ arms, shoulders | +++ eyes, arms | + | ++ | ++ – +++ | Small, if care taken |
| Backpacking | 3-8 | 250-600 | ++ walking<br>+++ climbing | ++ | + | + – +++ | + | +++ | Sprains, back strain |
| Badminton | | | | | | | | | |
| Social doubles | 3-4 | 250-300 | + | + | +++ | +++ | ++ | ++++ | ++ |
| Social singles | 6 | 450 | ++ | + | +++ | +++ | ++ | +++ | ± |
| Compet. singles | 8-10 | 600-750 | +++ – ++++ | ++ | ++++ | ++++ | +++ | ++ | ± |
| Baseball or softball | | | | | | | | | |
| Except pitcher | 2-3 | 150-250 | ± | + | + | + | Stressful waiting | +++ | Hit by ball; bat; sliding trauma; sprains |
| Pitcher | 0 | 450 | ++ | ++ | +++ | ++ | ++ | +++ | |
| Basketball | 4-10 | 300-750 | ++++ | +++ | ++++ | ++++ | +++ | +++ | Collision injuries; knees, ankles, back |
| Bicycling (on level) | | | | | | | | | |
| 5 mph | 3 | 250 | + | ± legs | ++ | ± | stressful in traffic; otherwise +++ | +++ | Traffic hazards and spills |
| 10 mph | 6 | 450 | ++ | ++ | ++ | ± | | ++ | |
| 13 mph | 9 | 650 | +++ – ++++ | +++ | ++ | + | | + | |
| Boardsailing | 3-8 | 250-600 | ++ – +++ | ++ – ++++ | +++ – ++++ | +++ | +++ | ++ | Bruises; hypothermia |
| Bowling | 1½-3 | 100-225 | little | little | ++ | ++ | ± | ++++ | Low |
| Calisthenics | 2-8 | 150-600 | ++ – ++++ | + for most<br>+++ for some | ++ – +++ | ++++ if varied | ++ | ++ | Few, with good warm-up |
| Canoeing | | | | | | | | | |
| Flat water | 2-8 | 150-600 | ++ – +++ | +++ | ++ | ++ | +++ | +++ | Drowning; |
| White water | 5-10 | 400-750 | +++ – ++++ | ++++ | ++++ | ++++ | ++++ | ++ – ++++ | Hypothermia |
| Dancing | | | | | | | | | |
| Ballet & Modern | 4-9+ | 300-700+ | +++ – ++++ | ++ – ++++ | ++++ | ++++ | +++ | +++ | Sprains; tears; strains |
| Vigorous Ballroom | 3-8+ | 250-600 | ++ – +++ | ++ | +++ | +++ | +++ if relaxed | ++++ | Few |
| Folk & Square | 3-8+ | 250-600+ | ++ – +++ | +++ | +++ | ++ | +++ if relaxed | ++++ | Few |
| "Aerobic" | 5-9 | 300-700 | +++ – ++++ | +++ | +++ – ++++ | +++ – ++++ | ++++ | ++++ men still reluctant | Rare |
| Fencing | 6-9 | 450-700 | +++ | +++ | ++++ | ++++ | +++ | ++ | Considerable, if guards defective |

**Copy This Page for Patient Information**

| Activity or Exercise | METs[1] | KCAL per h[2] | Cardio-respiratory Endurance | Muscle Strength, Power & Endurance | Coordination, Balance & Grace | Flexibility & Agility | Stress Management/[3] Diversion | Social Oppor-tunities | Injury & Hazard Problems[4] |
|---|---|---|---|---|---|---|---|---|---|
| Fishing | | | | | | | | | |
| Casting | 2-3 | 150-250 | little | – | + | ++ | +++ | ++ | Fish hook in eyes; drowning; and hypothermia |
| Walking with waders | 4-6 | 300-500 | ++ | + | ++ | ++ | +++ | ++ | |
| Football (while active) | 6-9 | 450-700 | ++ | +++ | ++ | +++ | Diverting | +++ | +++ |
| Gardening | 2-8 | 150-600 | ++ | ++ | + | ++ | +++ | ++ | Few |
| Golf | 2-4 | 150-300 | ++ walking | ± | ++ highly specific | +++ highly specific | ++ if relaxed | +++ – ++++ | Hit by ball or club |
| Gymnastics | 3-5 | 250-400 | ++ – ++++ | +++ – ++++ | ++++ | ++++ | +++ | ++ | Occasional |
| Handball | 6-10 | 450-750 | +++ – ++++ | +++ | ++++ | +++ | +++ | ++ | Sprains; muscle tear |
| Hockey | | | | | | | | | |
| Field | 8-10 | 600-750 | ++++ | ++ | ++ | +++ | +++ | +++ | Some |
| Ice | 8-10 | 600-750 | +++ | ++ | ++ | +++ | +++ | +++ | More |
| Isometrics | 2-5 | 150-400 | + | up to ++++ | little | + – ++ | ± | + | Few |
| Isotonics | 2-10+ | 150-800 | +++ | ++ | ++ | +++ | ++ – +++ | ++ | Few |
| Jogging (see Running) | | | | | | | | | |
| Karate/Judo | 6-10+ | 450-800+ | +++ – ++++ | ++ – +++ | ++++ | ++++ | Diverting & stressful | up to ++++ | Contact injuries |
| Kayaking (see Canoeing) | | | | | | | | | |
| Mountain-climbing | 6-8 | 450-800 | +++ | +++ – ++++ | ++ | +++ | Can be stressful | ++ | Falling rocks; avalanches |
| Mowing | | | | | | | | | |
| Pushing Power | 3-4 | 250-300 | + | + | little | little | little | little | Considerable |
| Pushing Hand | 6-8 | 450-600 | +++ | +++ | little | little | little | little | Few |
| Paddleball/ Platform Tennis | 4-8 | 300-600 | +++ – ++++ | ++ | +++ | ++++ | +++ | +++ | Eye injury |
| Ping Pong (Table Tennis) | 4-6 | 300-450 | +++ | + | +++ | +++ | +++ | +++ | Few |
| Racquetball | 6-10 | 450-750 | ++++ | ++ | +++ | ++++ | +++ | +++ | Eye, racquet injuries |
| Raking Leaves | 3-5 | 250-400 | ++ | + | + | + | ++ | + | Few |
| Riding Horseback | 2-5 | 150-400 | + – +++ | + – ++ | ++ | ++ | ++ | ++ | ++ |
| Rope Skipping | 8-12 | 600-900 | ++++ | +++ | +++ | +++ | +++ | ++ | Few |
| Rowing | | | | | | | | | |
| 2 mph | 3 | 250 | + | ++ | ++ | ++ | +++ | +++ | Upsets; hypothermia |
| 4 mph | 7 | 500 | ++ | +++ | ++ | ++ | +++ | + | |
| 6 mph | 12 | 900 | ++++ | ++++ | ++ | ++ | +++ | – | |
| Rugby | 6-8 | 450-600 | +++ | +++ | + | ++ | ++ | +++ | Lacerations; bruises |
| Running & Jogging | | | | | | | | | |
| 5 mph | 7-8 | 500-600 | +++ | ++ | little | little | + | ++ | Hit by autos; sprains |
| 7 mph | 12 | 800 | ++++ | +++ | little | little | ++ | + | |
| 9 mph | 15 | 1100 | ++++ | ++++ | little | little | + | + | |

**Copy This Page for Patient Information**

| Activity or Exercise | METs[1] | KCAL per h[2] | Cardio-respiratory Endurance | Muscle Strength, Power & Endurance | Coordination, Balance & Grace | Flexibility & Agility | Stress Management/[3] Diversion | Social Oppor-tunities | Injury & Hazard Problems[4] |
|---|---|---|---|---|---|---|---|---|---|
| Sailing | | | | | | | | | |
| Crew | 2-4 | 150-300 | ++ | +++ | +++ | +++ | +++ | +++ | Overboard hazards |
| Skipper | 1-3 | 75-200 | – | + | + | + | ++ | ++ | |
| Hand Sawing Hardwood | 6-8 | 450-600 | +++ | +++ | little | little | + | ± | Cuts |
| Sexual Intercourse | 5-8 | 400-600 | ....Usually too brief.... | | unlikely | ++ | ++ – ++++ | ? | Too macho or "conquest" coronary |
| Shovelling | 5-9 | 400-700 | ++ – ++++ | +++ – ++++ | + | ++ | +++ | ± | Backstrain |
| Skating | 4-10+ | 300-800+ | ++ – ++++ | ++ – ++++ | ++++ | +++ | +++ | ++++ | Falls; cuts |
| Skiing | | | | | | | | | |
| Cross country | 5-12+ | 400-900+ | ++ – ++++ | ++ – ++++ | +++ | +++ | ++++ | +++ | Few strains, fractures |
| Downhill | 4-10+ | 300-800+ | ++ – +++ | ++ – ++++ | ++++ | ++++ | ++++ | +++ | |
| Skin diving | 6-10 | 450-750 | +++ | +++ | + | ++ | +++ | + | Drowning; sharks; hypothermia |
| Soccer | 8-10+ | 600-750+ | +++ – ++++ | +++ | ++++ | +++ | +++ | ++ | Collisions |
| Squash | 8-10+ | 600-750 | ++++ | +++ | ++++ | ++++ | ++++ | ++ | Eye injuries; collisions |
| Surfing | 4-7 | 300-500 | ++ | +++ | ++++ | ++++ | +++ | ++ | Coral cuts |
| Swimming | 4-10+ | 300-750 | ++++ | +++ | ++ | ++ | ++++ one of best | +++ | Drowning; hypothermia |
| Tennis | 4-10 | 300-750 | ++ – ++++ | +++ | ++++ | +++ | +++ | ++++ | Eye injuries; sunburn; sprains |
| Volleyball | 4-7 | 300-500 | ++ – +++ | ++ | +++ | +++ | ++ | ++++ | Sprains; collisions |
| Walking (on level) | | | | | | | | | |
| 2 mph 3 kph | 2 | 150 | + | ± | ± | little | ++ | +++ | Minimal |
| 3 mph 5 kph | 3+ | 250 | ++ | + | + | little | +++ | ++ | Minimal |
| 4 mph 6.5 kph | 5-6 | 400-500 | +++ | ++ | ++ | ++ | +++ | + | Low |
| (stairs/hills) | 7-12+ | 500-900 | +++ | +++ | + | ++ | ++ | ++ | Low |
| Waterskiing | 4-8 | 300-600 | + – ++ | +++ | ++ | ++ | exciting | ++ | Propeller cuts; back strain |
| Weight lifting | 3-6 | 250-450 | + | ++++ | ++ | + | stressful | + | High blood pressure; dropped weights |
| Windsurfing (see Boardsailing) | | | | | | | | | |
| Wood splitting | 2-6+ | 150-500 | ++ – +++ | +++ | +++ | ++ | +++ | + | Eye and foot injuries |
| Yoga | 1-4 | 75-300 | little | little | variable | up to ++++ | may be excellent | +++ | Few |

---

* 1) METs = Multiples of resting metabolic rate—when sitting. The range reflects the varying intensity, from the leisurely or recreational pace to the competitive or frenetic, that can be involved.

2) Kilocalories per hour—based on a 70 kg (154 lbs) weight. A 10% increase or decrease can be applied for each 7 kg (15 lbs) over or under 70 kg, respectively.

3) Stress management may operate by either of at least two mechanisms or both: a) Through diversion, which makes it difficult to continue concerns about previous worries; b) through high level metabolic and muscular activity, which reduce chronic muscle tension and enhance sleep, rest and relaxation. Some activities such as karate and waterskiing may be "stressful" due to the excitement involved, but provide good recovery relaxation.

4) Injury & Hazard. Most hazards can be avoided with care and appropriate stretching; warming up and cooling down can help reduce strains, tears, and joint irritations that otherwise make some activities painful.

* usually approximately 3.5 ml/kg/min $O_2$ consumption

**Copy This Page for Patient Information**

# Appendix C

### How To Stretch

Stretching is easy to learn. But there is a right way and a wrong way to stretch. The right way is a relaxed, sustained stretch with your attention focused on the muscles being stretched. The wrong way (unfortunately practiced by many people) is to bounce up and down, or to stretch to the point of pain: these methods can actually do more harm than good.

If you stretch correctly and regularly, you will find that every movement you make becomes easier. It will take time to loosen up tight muscles or muscle groups, but time is quickly forgotten when you start to feel good.

### The Easy Stretch

When you begin a stretch, spend 10 to 30 seconds in the *easy stretch*. No bouncing! Go to the point where you feel a *mild tension*, and relax as you hold the stretch. The feeling of tension should subside as you hold the position. If it does not, ease off slightly and find a degree of tension that is comfortable. The easy stretch reduces muscular tightness and readies the tissues for the developmental stretch.

### The Developmental Stretch

After the easy stretch, move slowly into the *developmental stretch*. Again, no bouncing. Move a fraction of an inch further until you again feel a mild tension and hold for 10 to 30 seconds. Be in control. The tension should diminish; if not, ease off slightly. The developmental stretch fine-tunes the muscles and increases flexibility.

### Breathing

Your breathing should be slow, rhythmic and under control. If you are bending forward to do a stretch, exhale and then breathe slowly as you hold the stretch. Do not hold your breath while stretching. If a position inhibits your natural breathing pattern, then you are obviously not relaxed. Just ease up on the stretch so you can breathe naturally.

### Counting

At first, silently count the seconds for each stretch. This will insure that you hold the proper tension for a long enough time. After a while, you will be stretching by the way it feels, without the distraction of counting.

### The Stretch Reflex

Your muscles are protected by a mechanism called the *stretch reflex*. Any time you stretch the muscle fibers too far (either by bouncing or overstretching), a nerve reflex responds by sending a signal to the muscles to contract; this keeps the muscles from being injured. Therefore, when you stretch too far, you tighten the very muscles you are trying to stretch! (You get a similar involuntary muscle reaction when you accidentally touch something hot; before you can think about it, your body quickly moves away from the heat.)

Holding a stretch as far as you can go or bouncing up and down strains the muscles and activates the stretch reflex. These harmful methods cause pain, as well as physical damage due to the microscopic tearing of muscle fibers. This tearing leads to the formation of scar tissue in the muscles, with a gradual loss of elasticity. The muscles become tight and sore. How can you get enthused about daily stretching and exercise when these potentially injurious methods are used?

Many of us were conditioned in high school to the idea of "no gain without pain." We learned to associate pain with physical improvement, and were taught that "the more it hurts, the more you get out of it." But don't be fooled. Stretching, when done correctly, is not painful. Learn to pay attention to your body, for pain is an indication that something is *wrong*.

The easy and developmental stretches, as described on the previous page do not activate the stretch reflex and do not cause pain.

This diagram will give you an idea of a "good stretch":

<table>
<tr><th colspan="3">← A STRETCH →</th></tr>
<tr><td>An Easy Stretch<br>*(hold for 20 to 30 seconds)*</td><td>The Developmental Part of Stretching<br>*(hold for 30 seconds or longer)*</td><td>A Drastic Stretch<br>*(Do not extend a stretch until you feel pain)*</td></tr>
</table>

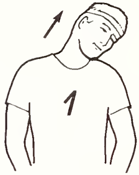
The straight line diagram represents the stretch which is possible with your muscles and their connective tissue. You will find that your flexibility will naturally increase when you stretch, first in the easy, then in the developmental phase. By regularly stretching with comfortable and painless efforts you will be able to go beyond your present limits and come closer to your personal potential.

Excerpted from Anderson.[29]

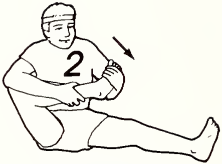
**Copy This Page for Patient Information**

## *Cycling Stretches*

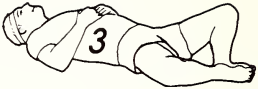
*(Before and after cycling)*
*Approximately 10 minutes*



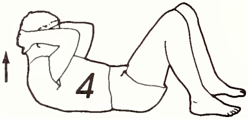
5 times
each direction



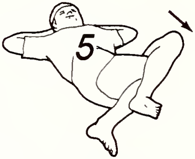
10 times
each direction



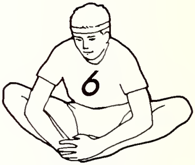
30 seconds



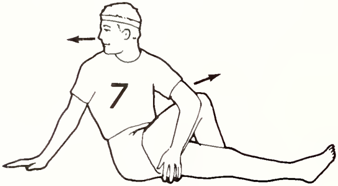
3 times
5 seconds each



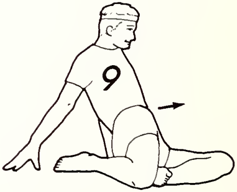
30 seconds
each side



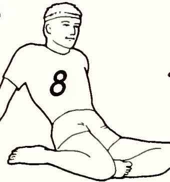
30 seconds



15 seconds
each side

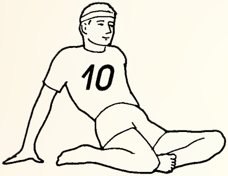


5 seconds

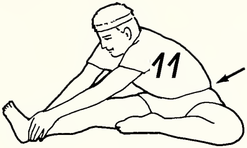


20 seconds

**Copy This Page for Patient Information**



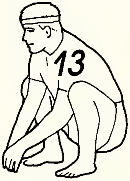
*20 seconds*



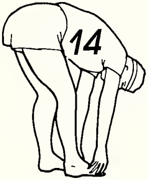
*30 seconds*

**12**

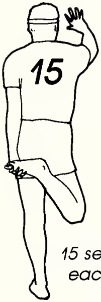
*Repeat*
*8, 9, 10, 11*
*other leg*



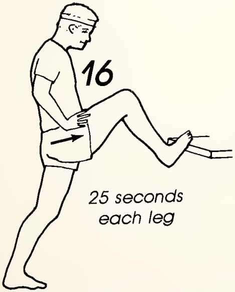
*20 seconds*



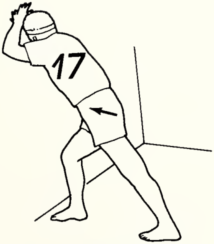
*30 seconds*



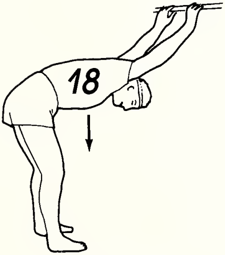
*15 seconds*
*each leg*



*25 seconds*
*each leg*



*30 seconds*
*each leg*



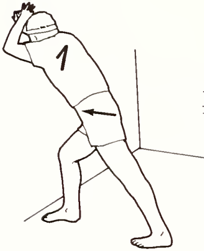
*30 seconds*

**Copy This Page for Patient Information**

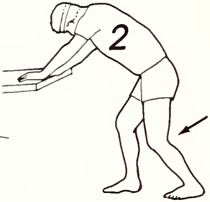
## *Walking Stretches*

*(Before and after walking)*
*Approximately 7 minutes*

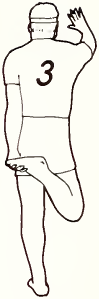
*These stretches will make the movements of walking feel free and easy.*



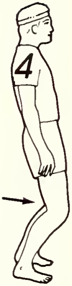
*30 seconds each leg*



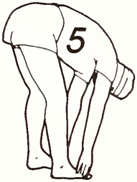
*20 seconds each leg*



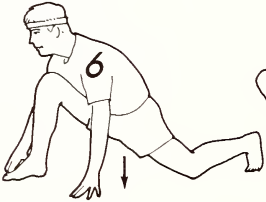
*15 seconds each leg*



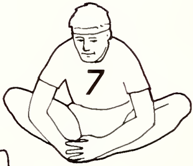
*30 seconds*



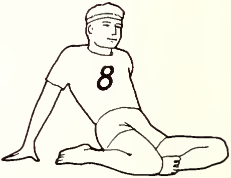
*30 seconds*



*20 seconds each leg*

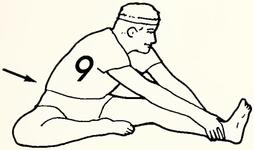


*30 seconds*

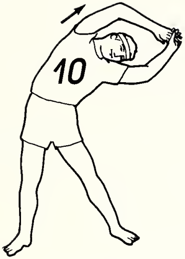


*20 seconds each leg*

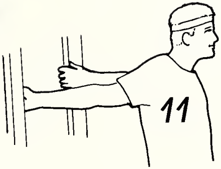
**Copy This Page for Patient Information**



*20 seconds each leg*



*8 seconds each side*



*30 seconds*

**Copy This Page for Patient Information**

## *Over 50 Stretching*

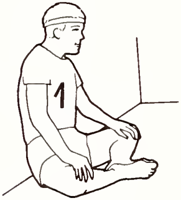
*Approximately 5-6 minutes*

It is never too late to start stretching. In fact, the older we get, the more important it becomes to stretch on a regular basis.

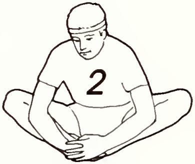
With age and inactivity, the body gradually loses its range of motion; muscles can lose their elasticity and become weak and tight. But the body has an amazing capacity for the recovery of lost flexibility and strength if a regular program of fitness is followed.

The basic method of stretching is the same regardless of differences in age and flexibility. *Stretching properly means that you do not go beyond your own comfortable limits.* You don't have to try to imitate the drawings in this book. Learn to stretch your body without force; stretch by how you feel. It will take time to loosen up tight muscle groups that have been this way for years, but it can be done with patience and regularity. If you have any doubts about what you should be doing, consult your physician *before you start.*

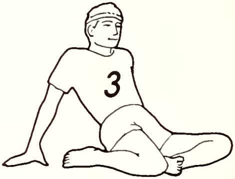
Here is a series of stretches to help restore and maintain flexibility.



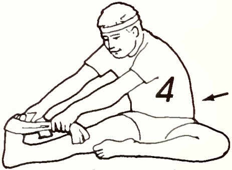
*20 seconds*



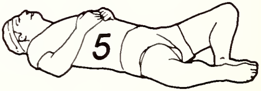
*30 seconds*



*20 seconds*
*each leg*

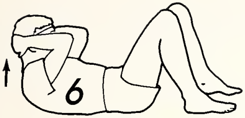


*20 seconds*
*each leg*

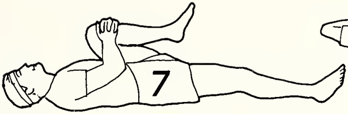


*30 seconds*

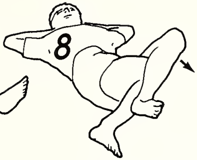
**Copy This Page for Patient Information**


6

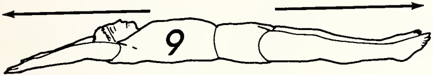
5 seconds each
3 times


7

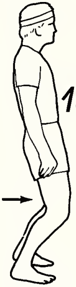
20 seconds
each side


8

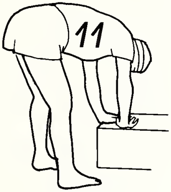
20 seconds
each side


9

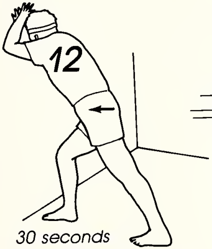
3 times
5 seconds each


10

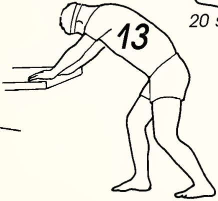
20 seconds


11

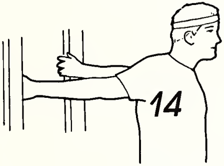
20 seconds


12

30 seconds
each leg

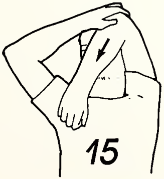

13

15 seconds
each leg

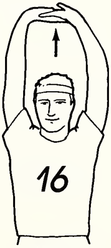

14

15 seconds

**Copy This Page for
Patient Information**


15

10 seconds
each arm


16

10 seconds

Adapted from *Stretching*,
by Bob Anderson,
Shelter Publications,
Bolinas, CA 1980.

# Health Education

**Helen Mellett, RN, Dr Ph**
**Adult Nurse Practitioner**
**Assistant Professor of Nursing**
**Columbia School of Nursing**
**New York, NY**

Two basic premises underlie the philosophy and framework of the following discussion on the educational component of diabetes management. The first is that the traditional health-care system is still rooted in acute care. As a result, linking education and preventive measures to the distant outcomes of chronic illnesses such as diabetes is problematic in the organization of health-care delivery. For example, the time clinicians spend on psychosocial issues important in diabetes management is, for the most part, not reimbursable. Yet, an increasing amount of evidence suggests that health education can be an effective component of diabetes management.[1] Even so, there are few incentives for providers to incorporate health education into their management plans.

The second premise assumes that self-care is as important as clinical care, because, in many chronic illnesses, most interventions take place outside of an institutional setting. This is particularly true with diabetes, for which the major focus of intervention is on diet. There is no escape from it; everyone has to eat.[2]

Given these premises, our discussion is framed around the model of the patient's visit to the provider. At this time, the clinician is directly involved in delivering health services, including education, to the diabetic patient. This is not to negate or minimize the contribution made by formal group education programs. The first purpose is to reaffirm the interventional role of education in primary care. The second is to underscore the importance of psychosocial issues in educational concepts.

Routine care for the NIDDM patient usually starts with diagnosis, commonly as a result of tests that confirm what has already been suggested by presenting signs or symptoms. The clinician brings to this initial encounter a set of expectations indicating that the initial goal of NIDDM treatment is to normalize glucose, usually starting with regulating weight and, sometimes, proceeding to the prescription of hypoglycemic agents. Another set of expectations has to do with secondary prevention—interventions to prevent patients from developing diabetic complications or, when this is not possible, to minimize them through early interventions and treatment.[3]

## A Psychosocial Framework

While these expectations may appear self-evident, they raise a host of psychosocial issues. Every knowledgeable clinician knows that activities designed to manage health problems should stem from a data base. The framework for the data is well known, including history of the present illness, review of systems, and physical exam. For clinicians, who assemble this important information, the following questions, suggested by Blainey, provide a psychosocial framework for determining a patient's level of understanding and for spotting difficulties a patient may have in accommodating his lifestyle to the new realities of diabetes:

)How has diabetes affected your body?

- What effect do you believe diabetes will have on your body over time?
- What do you believe caused your diabetes?
- What do you believe you can do to minimize the effects of diabetes?
- Where does treating your diabetes rank in terms of personal goals?[2]

Although certain information must be obtained during the initial visit, it is almost impossible to compile all the appropriate psychosocial data in such a brief time; few people are comfortable discussing their problems with strangers. At least three visits are required to establish a trusting relationship with patients, and to develop mutual expectations about therapy. The questions "How are things at home? Is your spouse supportive?" will evoke more honest responses after patients get to know the provider. Consequently, a major dimension of psychosocial issues is the establishment of a good provider-patient relationship in which education can take place.

A second dimension is the very nature of chronic illnesses. The role of family and community is important to everyone, but it takes on added significance in chronic illnesses where intervention takes place outside of institutional settings. Obviously, those diabetics with good family and support systems will do better than those without. The provider's job is to determine whether support systems are adequate and make referrals to community agencies when appropriate.

### The Aims of Education

Specific examples drawn from a hypothetical data base can illustrate how to pinpoint opportunities for health education. Incidence and prevalence data make it clear that many newly diagnosed NIDDM patients are overweight, smoke cigarettes, and have coexisting hypertension.[3,4] Again, the well-informed clinician knows that the best way for patients to gain self-management skills for any chronic illness is through teaching and counseling, which must be offered by the primary care provider during an office visit.[5] To be effective, such education must transmit knowledge and skills, continually reinforce positive behaviors, and ensure support from the physician, nurse practitioner, other staff members, and peers.[6]

It is well known that behavior-oriented patient education is generally more powerful than didactically delivered information or programs. The essential feature of proper diabetes education is an emphasis on behaviors that enhance self-care, not on the disease process itself.[1]

Given all this, we can consider the hypothetical NIDDM patient—recently diagnosed, overweight, hypertensive, and a smoker. Our task is to implement intervention strategies to normalize his weight, blood glucose level, and blood pressure, and to stop him from smoking—a tall order, by any standard.

For most Type II patients, the initial approach to normalizing glucose levels is to normalize weight. However, some caveats should be mentioned. Except for certain self-selecting populations, it is difficult, if not impossible, for overweight persons to maintain weight loss. Most have thought about losing weight, and most have expended considerable effort trying to do so.

### Achieving Weight Loss

How the clinician introduces the idea of weight loss will have a significant bearing on the patient's success. Instead of setting weight reduction as an explicit goal, the clinician should point out that dietary measures are the most effective first step in normalizing blood glucose levels. At the same time, it should be acknowledged that this is a fairly difficult task for most people, including diabetics. Then, focusing on previous attempts and current possibilities, the clinician should discuss a dietary regimen in the context of the patient's lifestyle.

If a joint decision can be made to attempt weight reduction, the clinician needs to give the patient specific counseling on establishing a self-management plan. Basic to this is the advice that joining a group usually works better than attempting management alone. And because excess weight is gained over a long period of time, the patient should be told that losing it the same way, one and one half to two pounds a week, is a reasonable goal. Hospitalization is an alternative approach. While this can offer a reasonable beginning for behavior modification, and an opportunity for closely monitored fasting, it seldom is reimbursable by third-party payers.

In a weight-reduction agreement, as with any contract, clinicians must neutralize their own expectations. Put another way: Don't get into power struggles with patients. The goal is the patient's; the provider's task is to give authoritative information about choices and methods to implement short-term objectives and long-term goals. If the short-term objective of weight reduction is not achieved, alternative treatment must be undertaken. The important point to remember is that the failure of early interventions to promote effective self-management does not abrogate the clinician's responsibilities: monitoring blood glucose levels, initiating medications to normalize those levels, and monitoring for diabetic complications. The importance of patients keeping appointments must be emphasized, even if a short-term objective, such as weight reduction, remains out of reach.

In this first encounter, the issue of smoking can't be ignored. But because it is unreasonable to expect a patient to change two behaviors simultaneously, it is necessary to choose between weight reduction and this unhealthy habit. When the risk of macrovascular complications is high, smoking cessation takes priority. If weight reduction is considered paramount, the issue of smoking can be tabled for later discussion.

Patients come to the first encounter with their own expectations. Adjustment to diabetes can be complicated by the absence of physical symptoms. This often makes it difficult for patients to summon motivation in accepting self-management strategies that can normalize glucose and lessen the risk of complications.[2] At the other extreme, many patients have distorted perceptions of what the future holds. Grim details about the toll the illness takes may be part of their family lore. As a result, patients often stratify their concept of the disease into unrealistic grades of severity; in the eyes of many patients, NIDDM is not associated with long-term complications.

### Attention to Details

Routine health care for diabetic patients is similar to that for nondiabetic ones. Follow-up visits should include the interval history, physical examination, assessment, and the treatment plan. The plan includes five components: diagnostic testing, therapeutic measures, patient education, referrals, and return visits. The clinician who provides routine care knows that there has been a steady decline in deaths of diabetic patients from diabetic ketoacidosis and infection, but a rise in deaths from cardiovascular and renal complications.[4] Yet, even though hypertension is twice as prevalent among diabetics, only 75 percent of diabetic patients with hypertension are being treated for the condition. In fact, clinicians check diabetic patients' blood pressure only two thirds of the time.[3] Given this and the absence of hard data, we can only speculate about how frequently they examine the feet of diabetic patients. And given the fact that diabetic retinopathy is a leading, but preventable, cause of blindness, it's hard to understand why fewer than half of all diabetics are examined each year for this complication.[3,7]

The goal in stating these facts is not to chide clinicians, but to point out how these omissions are construed by patients. Assuming, for example, that control of hypertension and good foot care have been emphasized as important, many diabetic patients have trouble understanding why clinicians seem to lose interest in monitoring these variables as time goes on. Clinicians who pay assiduous attention to monitoring weight, self-testing, hypertension, and foot care offer positive reinforcement of patients' self-management behaviors. Conversely, failure to monitor implies self-management does not matter.

Even though routine health care may be similar for diabetics and the general population, the clear risk of diabetic complications should demand emphasis on aspects of health maintenance for diabetic patients. Because diabetes is the leading cause of blindness in adults, periodic eye exams are essential to monitor for retinopathy, glaucoma, and cataracts. Dental care is also a high priority since plaque accumulates more readily and periodontal disease progresses more rapidly in diabetics. And because diabetics are at high risk for influenza and pneumonia, pneumococcal vaccine and yearly influenza vaccine should be encouraged.

### Toward Healthier Lifestyles

Returning to the issue of the diabetic smoker, it is clear that smoking is a major public health problem by itself. But it is especially dangerous for diabetics because it accelerates the onset of macrovascular complications. It is the clinician's responsibility to show how smoking is relevant to the diabetic patient's health. In doing this, the clinician faces the difficult task of applying population-based data to the individual diabetic patient at risk. We know, for example, that people with diabetes are twice as likely to die of heart disease than is the general population. We know that ischemic heart disease is implicated in 60 percent of deaths of adults with diabetes.[4] But what the patient wants to know is whether stopping smoking will significantly reduce risk. This cannot be answered with certainty; we can speak only in terms of probability.

Bass and Grantham suggest that when working with any population that follows an unhealthy lifestyle, it may be useful to partition the population.[8] They divide the population into four categories:

- "changers"—those who change their behavior independently and need no external support
- "conditional changers"—those who change their behavior when given the appropriate tools
- "doubting nonchangers"—those who do not believe themselves to be interested in changing their behavior but wish to learn more about the risks and the process of change
- "loyal nonchangers"—those who are neither interested in reducing their risk nor in changing their unhealthy behavior.

There is no easy guide to this categorization, but the history should give the clinician an indication of where a patient best fits. Statistics suggest that 75 percent of smokers are either "conditional changers" or "doubting nonchangers," and that strategies for intervention should be aimed at these populations. An additional 20 percent of smokers are thought to be "loyal nonchangers," for whom no intervention strategies are warranted. These dedicated smokers have decided to accept the risks associated with their addiction.[8] With friendly persuasion and skillful use of authoritative information, the clinician might be able to shift "loyal nonchangers" into a category more amenable to intervention. A rabid antismoking campaign against diabetic smokers in this group is almost sure to alienate patients and, probably, cause patient attrition.

So far, the discussion has focused on aspects of provider-patient interaction that affect educational intervention strategies. My closing comment is that NIDDM is a chronic illness whose clinical presentation often obscures the possibility of serious morbidity and early mortality. Adjustment to diabetes is often complicated by a lack of visible physical evidence of the disease. This can reinforce the patient's ability to deny important problems.[9]

How well patients with diabetes respond to health education and self-care is a complex process involving beliefs and expectations—on the parts of both patients and providers. Education during clinic visits can make it easier for patients to understand that they face a disproportionate number of risks. Education in the clinical setting can also encourage them along the way to healthier and better lives.[3]

## References

1. Mazzuca SA, Moorman NH, Wheeler ML, et al: The Diabetes Education Study: A controlled trial of the effects of diabetes patient education. *Diabetes Care* 1986;9:1-9.
2. Blainey, CA: Diabetes mellitus, in Patrick M, et al [eds]: *Medical-Surgical Nursing: Pathophysiological Concepts.* New York, JB Lippincott Co, 1986, pp 1028-1052.
3. The Carter Center of Emory University: Closing the gap: The problem of diabetes mellitus in the United States. *Diabetes Care* 1985;8:391-406.
4. Brunner L, Suddarth D: *Textbook of Medical Surgical Nursing.* New York, JB Lippincott Co, 1988, pp 903-922.
5. Phipps WJ, Long BC, Woods NF. *Medical-Surgical Nursing: Concepts and Clinical Practice.* St. Louis, CV Mosby Co, 1987, p 627.
6. Anderson RM: Defining and evaluating diabetes patient education. *Diabetes Care* 1983;6:619-620.
7. Collins JW: Proposed criteria for referring diabetic neuropathy. *The Nurse Practitioner* 1983;13:21-25.
8. Bass F, Grantham RP: Preventive medicine in Rakel RE, Conn HF (eds): *Family Practice.* Philadelphia, WB Saunders Co, 1978.
9. Patrick M, et al (eds): *Medical-Surgical Nursing: Pathophysiological Concepts.* New York, JB Lippincott Co, 1986, p 1050.

# Diabetes Education and Behavior Modification

**Jane Darling, RN, ANP, CDE**
**Diabetes Education Coordinator**
**Mount Sinai Hospital**
**Hartford, CT**

Diabetes management is unique in that it requires the active participation of a knowledgeable patient to achieve permanent behavior changes. Motivating and educating these patients is a challenge that health-care professionals face daily.[1]

One in 20 Americans lives with the personal and economic costs of diabetes; 90 percent have Type II or non-insulin-dependent diabetes mellitus (NIDDM), while the remainder have Type I or insulin-dependent diabetes mellitus (IDDM).

The economic costs of diabetes—over $10 billion per year—result from a multitude of complications (Figure 1). Acute complications include hypoglycemia, diabetic ketoacidosis, and hyperglycemic hyperosmolar nonketotic coma. Long-standing diabetes can lead to eye diseases that impair vision and cause blindness, vascular disorders that result in heart disease and stroke, kidney disease that requires dialysis or transplantation, and nerve damage that causes peripheral and autonomic neuropathies. Yet, with successful patient education, these complications, and their personal toll, are not inevitable. Nor is premature mortality.

Better diabetes management can slow the development of complications. Studies that demonstrate this also point to the economic benefit of diabetes education: reduced hospitalization, fewer emergency room visits, and decreased medical care for long-term complications.[2] People with diabetes who understand their disease and know how to perform optimum self-care are better equipped to make lasting lifestyle changes. The diabetes educator can help patients break the "cycle of diabetes": Poor compliance leads to more severe disease and further reduces patient motivation (Figure 2).

The basic component of patient education is understanding what diabetes mellitus is and how it alters normal metabolism. Diabetes is characterized by fasting hyperglycemia or plasma glucose levels above defined limits (Table 1). It is a chronic disorder of carbohydrate, fat, and lipid metabolism (Figures 3, 4, and 5) resulting from a lack of insulin. Insulin is the hormone that regulates the blood glucose level and the uptake of glucose in the liver, muscle, and fat cells.

Specifically, with Type II diabetes, any of the following conditions may exist:

- Normal or increased basal insulin secretion
- Insulin resistance
- Tissue sensitivity to insulin and $\beta$-cell secretion in a state of flux
- Insulin resistance leading to a defect in insulin secretion, impaired $\beta$-cell function, and, ultimately, disturbances in insulin action[3]

These abnormalities are responsible for the classic symptoms of diabetes: polyuria, polyphagia, polydipsia, blurred vision, fatigue, frequent infection, and at times, weight loss (Table 2).

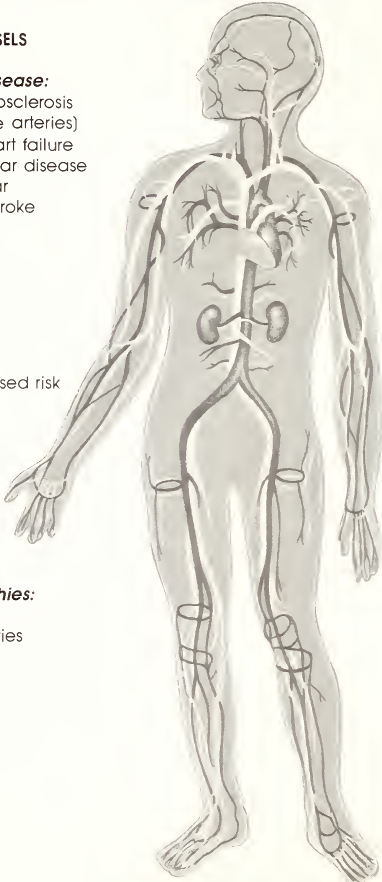
## Patient Education: The Key Component of Treatment

Effective diabetes education can help patients understand their condition, develop a positive at-

Figure 1

**What are the long-term complications associated with diabetes?**

Long-term complications can possibly be avoided with good control of blood sugar



**LARGE BLOOD VESSELS**

***Cardiovascular disease:***

Accelerated atherosclerosis
  (hardening of the arteries)
  Heart attack, heart failure
  Peripheral vascular disease
  Cerebral vascular
  insufficiency or stroke

***Problems with lower extremities:***

Peripheral vascular disease
Leg ulcers, gangrene, increased risk
  of infection

***Peripheral neuropathies:***

Nerve damage
Pain in lower extremities
Numbness

**SMALL BLOOD VESSELS**

***Visual complications:***

Retinopathy (hemorrhages
  in the retina of the eye,
  scar formation)
Impaired vision
Blindness

***Kidney disease:***

Massive protein loss
Renal failure—dialysis or
  transplantation

***Autonomic neuropathies:***

Impotence
Gastroparesis
Diabetic diarrhea
Neurogenic bladder
Impaired cardiovascular
  reflexes

***Foot problems:***

Poor sensation
Poor recovery
  from injury
Decreased sweating
Dry skin
Ulcerations
Infection
Gangrene

Figure 2

**It is up to *you* to break the cycle of diabetes!**

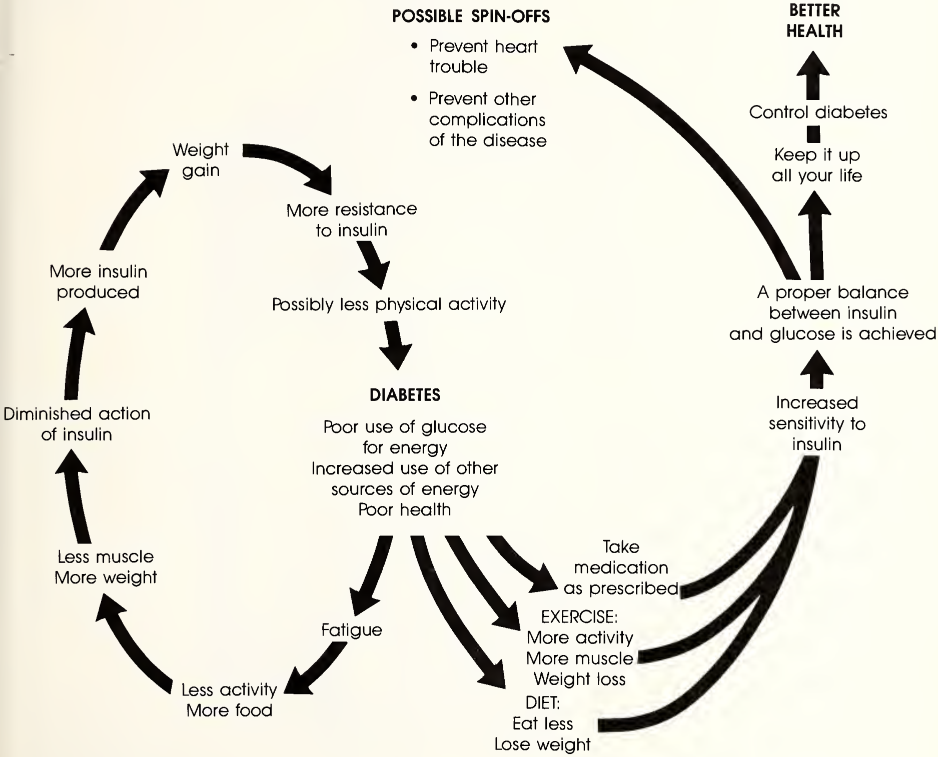

POSSIBLE SPIN-OFFS
• Prevent heart trouble
• Prevent other complications of the disease
BETTER HEALTH
Control diabetes
Keep it up all your life
Weight gain
More resistance to insulin
Possibly less physical activity
More insulin produced
A proper balance between insulin and glucose is achieved
DIABETES
Poor use of glucose for energy
Increased use of other sources of energy
Poor health
Diminished action of insulin
Increased sensitivity to insulin
Less muscle
More weight
Take medication as prescribed
EXERCISE:
More activity
More muscle
Weight loss
Fatigue
Less activity
More food
DIET:
Eat less
Lose weight

Table 1

**Diagnostic Criteria for Diabetes Mellitus**

- **Normal glucose tolerance:**
  Fasting blood sugar (FBS) <115 mg/dL *and...*
  2-hour postprandial <140 mg/dL *and...*
  No FBS or 2-hour postprandial value >200 mg/dL
- **Impaired glucose tolerance (after 75-g glucose load):**
  FBS <140 mg/dL *and...*
  2-hour postprandial >140 but <200 mg/dL *and...*
  At least one FBS value or 2-hour postprandial value >200 mg/dL
- **Diabetes mellitus:**
  FBS >140 mg/dL on at least two occasions *or...*
  Random glucose ⩾200 mg/dL with classic signs and symptoms of diabetes (polydipsia, polyphagia, polyuria, weight loss) *or...*
  FBS <140 mg/dL and 2-hour postprandial >200 mg/dL, with at least one value >200 mg/dL after 75-g glucose load

Adapted from criteria established by the National Diabetes Data Group of the National Institutes of Health.

titude, and acquire the self-management skills necessary to lead a productive and satisfying life.[4] The management of diabetes must focus on the prevention of poor control—a goal reached only by careful education.[5]

Although patient education has often been neglected, it is now recognized as a key component of quality health care and a patient right. Comprehensive education may well have the potential to increase the efficacy of the health-care delivery system. And increasingly, it is viewed as the legal responsibility of health-care professionals.[6-8] Diabetes educators must understand the crucial role they play in helping patients—and families—understand the disease and achieve effective management.

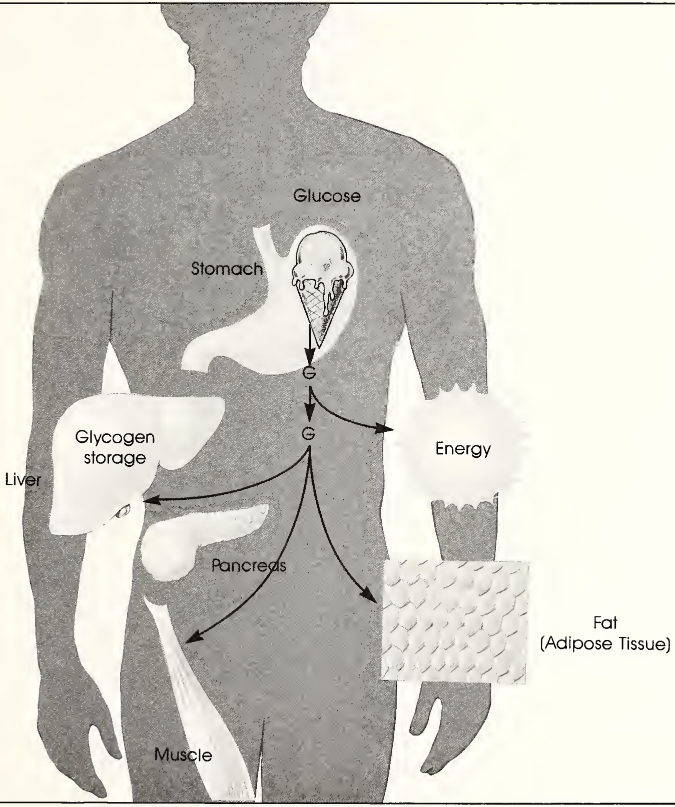
The importance of patient participation as well as individually tailored treatment plans cannot be overemphasized. Patients are more compliant when they know precisely what is expected of them. Research supports the premise that the more active the patient's participation, the more effective the learning experience will be.[6,9-11] The American Association of Diabetes Educators strongly advocates such an individual approach to patient education and management.[12]

Although diabetes education takes place in a variety of settings, ranging from one-on-one sessions to groups, this material is focused on health-care professionals providing one-on-one education in the office or outpatient setting.

There are some basic principles about how adults learn. When those principles are used as the foundation of diabetes education, they create the best chance of successful behavior changes. First, adults must be convinced that there is a problem they need to deal with and should express a readiness to learn. They need to be able to apply what

**CARBOHYDRATE**

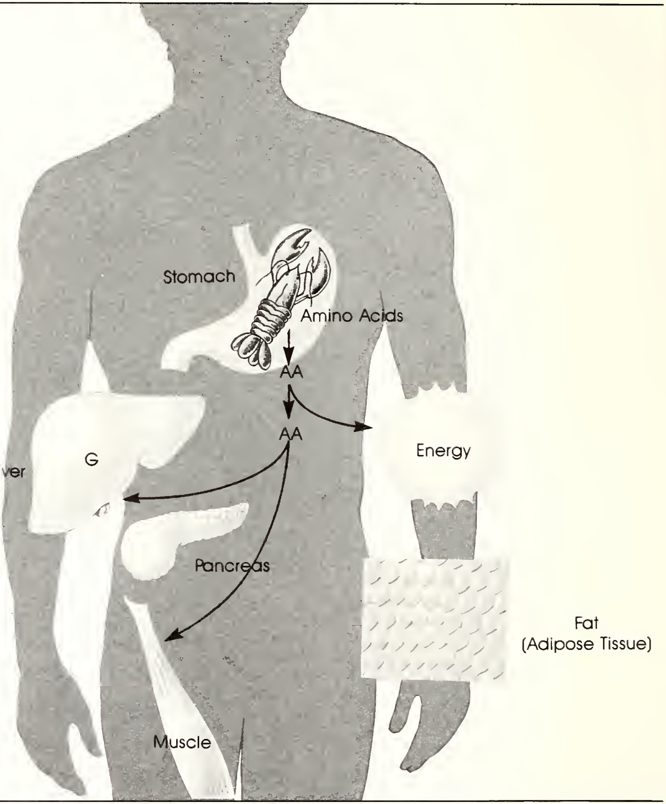
Figure 3



Carbohydrate, the world's most available—and the body's most usable—fuel, is found in sugars and starches. After such foods have been eaten, they are broken down into glucose. At that point, insulin allows the fuel to enter the cells, where it can be stored in the liver, the muscles, or in fat for future use.
(Adapted from Rossini A: The great diabetes machine. *Diabetes Forecast*, 29:22, 1976.)

## PROTEIN

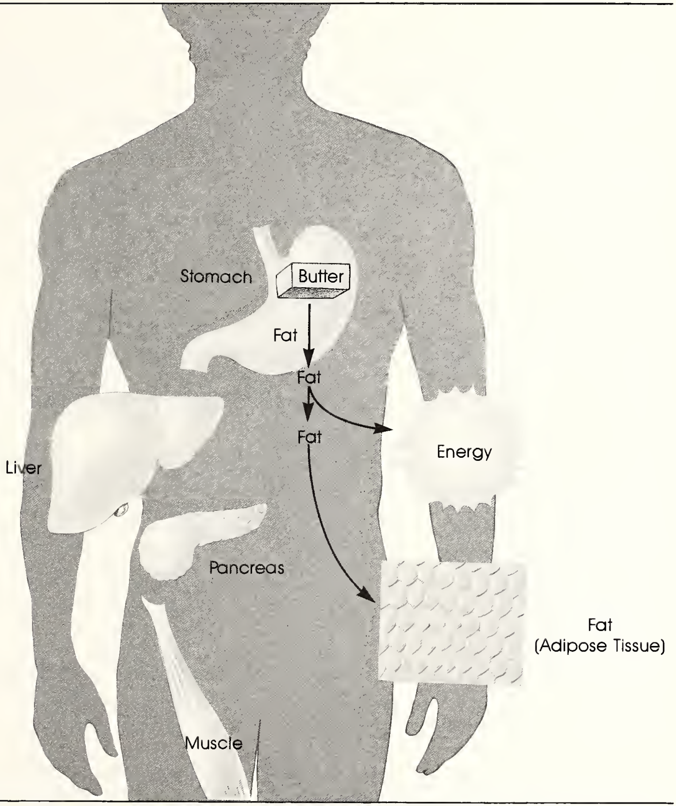
Figure 4



Sources of protein include dairy products, meat, and seafood. After ingestion, protein is broken down into amino acids, which—if insulin is present—supply energy for the body to use. Amino acids are used more slowly than glucose.
(Adapted from Rossini A: The great diabetes machine. *Diabetes Forecast*, 29.22, 1976.)

**FAT**

Figure 5



Fat, the body's third fuel source, is also less quickly available to the body than glucose. Sources of fat include meats, dairy products, and vegetable oils. As is true of protein and carbohydrate, the presence of insulin is required before fat's energy is available for use. As pictured above, some fat is stored in fat cells for future demand. Unless used, this energy source remains in storage.
(Adapted from Rossini A: The great diabetes machine. *Diabetes Forecast,* 29:22, 1976.)

Table 2

**Diabetes Mellitus: Classification and Characteristics**

| CLINICAL CLASSES | DISTINGUISHING CHARACTERISTICS |
|---|---|
| **Diabetes mellitus (DM)** | |
| **Type I Insulin-dependent diabetes mellitus (IDDM)** | Patients may be of any age, are usually thin, and usually have abrupt onset of signs and symptoms with insulinopenia before age 40. These patients often have strongly positive urine glucose and ketone tests, and are dependent upon insulin to prevent ketoacidosis and to sustain life. |
| **Type II Non-insulin-dependent diabetes mellitus (NIDDM) (obese or nonobese)** | Patients usually are older than 40 years at diagnosis, obese, and have relatively few classic symptoms. They are not prone to ketoacidosis except during periods of stress. Although not dependent upon exogenous insulin for survival, they may require it for stress-induced hyperglycemia and hyperglycemia that persists in spite of other therapy. |
| **Other types of diabetes mellitus** | Patients with other types of diabetes mellitus have certain associated conditions or syndromes. |
| **Impaired glucose tolerance (IGT) (obese or nonobese)** | Patients with impaired glucose tolerance have plasma glucose levels that are higher than normal but not diagnostic for diabetes mellitus. |
| **Other types of impaired glucose tolerance** | Patients with other types of impaired glucose tolerance have certain associated conditions or syndromes. |
| **Gestational diabetes mellitus (GDM)** | Patients with gestational diabetes mellitus have onset or discovery of glucose intolerance *during* pregnancy. |

Adapted from material in *The Physician's Guide to Type II Diabetes (NIDDM) Diagnosis and Treatment* (ADA, 1984).

they have learned, immediately, so that they internalize the new behavior.[13] After the educator demonstrates a new skill, having patients repeat the demonstration themselves is an effective way to apply learning. Educators must place the most emphasis on what patients feel they need to know—not what educators think patients need to know.

A patient's level of education is a key factor in determining the appropriate teaching methodology. The higher the education level, the less structure needed in teaching. This means using more complex and abstract methods. Conversely, with less formal education, more structure is needed in teaching. For example, less educated individuals need an itemized, concrete teaching plan that is simple and brief. Because the average American reads at a sixth-grade level, it is important to determine a patient's literacy level before selecting educational materials.[13]

Through previous learning experiences, people usually develop preferences for visual, audio, or tactile cues. The educator can identify the patient's preference by simply asking, "Tell me how you usually learn about something new." Structuring the teaching plan around this preference is possible because of the wide variety of teaching tools available. However, these tools do not replace the effectiveness of the teacher's demonstration followed by the student's imitation.

A "needs assessment" or a collection of information about the patient, is the first step in individualizing an educational program. This information should include the following: level of education and literacy, financial status, general health and health problems (especially visual acuity and mobility or any other deficits that may interfere with learning), support from family and friends, living arrangements, employment, cultural background, duration of diabetes, type of diabetes, family history of diabetes, previous diabetes education, present management plan, monitoring and control history, existing complications of diabetes, activity level, allergies to medication, recreational drug use, and dietary preferences. Most important, this information should be prioritized according to the patient perception of what he needs to know and not exclusively from the provider's perspective (see Figure 6, Assessment Record).

The actual teaching session is the second step in the educational process. Focusing on the patient's priorities first, the acronym DEEDS (based on a list developed by R. Keith Campbell, RPh) serves as an easy reminder of what the sessions should cover. DEEDS stands for:

- Diet—the cornerstone of diabetes treatment
- Education—the key to successful treatment
- Exercise—the forgotten prescription
- Drugs—the adjunct to therapy
- Self-monitoring—an indicator of the adequacy of treatment

Although every patient with Type II diabetes needs instruction in these basic areas, there are distinct levels of education—basic and advanced levels, as well as a maintenance or ongoing phase. All patients begin at the basic level and some may progress to the advanced level. Regardless of whether patients progress from one level to another, they all need continuing education.

Newly diagnosed patients, or those who are out of control, need short-term "survival-skill" instruction at frequent intervals. Together, patients and educators should identify responsibilities that will help patients participate immediately in self-care.

At the survival-skills level, based on behavioral objectives, patients should be able to:

- Verbalize their feelings about diabetes and their status (most important for the newly diagnosed, "How do you feel about having diabetes?")
- Define diabetes and what the disease does to the body
- Identify DEEDS treatment components
- Correctly demonstrate and perform self-monitoring techniques
- Identify personal goals of treatment
- Obtain identification bracelet or necklace
- Identify short-term problems (prevention, cause, treatment)
- Describe focal points of daily health care (skin,

Figure 6

## LIFESTYLE LESSONS ASSESSMENT RECORD

### Patient profile

last name first middle

address (street) city state zip

phone (home) (business)

date of birth

Sex M F

marital status S W M D

| | | | |
|---|---|---|---|
| Ethnic group: | Caucasian<br>Black | American Indian<br>Asian | Hispanic<br>other |

occupation medical insurance

alone family friends other

living arrangements

support system financial status

### Diabetes history

date of onset age at diagnosis

Type of diabetes

Type I ________
Type II (diet only) ________
(diet and oral agent) ________
(diet and insulin) ________
Other ______________________________

current symptoms

________ last blood glucose level

________ glycohemoglobin

____ / ____ cholesterol/triglycerides

parent(s) grandparent(s) sibling(s) twin children other
family incidence

________ prior diabetes education

Medical and health history

____ height ____ weight

Smoking:
never ____
quit ____
less than 1 pack ____
1 pack or more per day ____

________ allergies

________ visual acuity/color blindness

________ hearing acuity

Alcohol use:
never
1 to 2 drinks per week ____
3 to 10 per week ____
11 or more per week ____

________ dexterity

________ mobility

planning pregnancy ____
currently pregnant ____

Health problems

hypertension ____
cardiac ____
eye ____
vascular ____

infections ____
neuropathy ____
renal ____
dental ____

Medications for health problems

____________________________________________________

____________________________________________________

____________________________________________________

____________________________________________________

____________________________________________________

Present diabetes management

__________________________
meal plan

__________________________
oral agent

__________________________
insulin

Exercise pattern:

never ________
1 to 2 days per week ________
3 to 5 days per week ________
6 to 7 days per week ________

| Monitoring | Method | Frequency |
|---|---|---|
| Blood glucose | __________ | __________ |
| Urine glucose | __________ | __________ |
| Urine ketone | __________ | __________ |

| | | |
|---|---|---|
| Are you satisfied with your state of control? | yes | no |
| Are you willing to change your lifestyle? | yes | no |
| Are you aware of any diabetes complications? | yes | no |

### Nutritional history

previous diets ______________________________
recent weight change ______________________________
problem times and situations that affect eating habits ______________________________
______________________________

food allergies ______________________________
digestive problems ______________________________

vitamins/supplements used ______________________________

does own shopping yes no
does own cooking yes no

over desired body weight yes no
under desired body weight yes no

favorite foods ______________________________

eating pattern: 24-hour recall

| 1st meal | 2nd meal | 3rd meal | snacks |
|---|---|---|---|
| | | | |

### Potential educational barriers

highest level of education completed: ______________________________

native language: ______________________________

attitude toward diabetes: ______________________________
(patient's feelings)

### Immediate needs

(patient's perceived needs)

### Evaluation

### Follow-up

Adapted from Teza et al: Resource Manual to Help Your Program Meet National Standards, 1987.

feet, eyes, dentition)

- Develop individual sick-day rules
- Understand how to gain access to the health-care system
- Become aware of long-term complications
- Know available community-care services

Teaching is most effective if the educator proceeds very slowly and focuses on one or two concepts per session. Sessions can vary in length from 15 minutes to one hour, depending on the content and the patient's rate of learning. But in longer sessions, educators should be careful not to overload the patient with information. Patients who are overwhelmed may nod continually in agreement. A simple way of checking this is to say to the patient, "I just explained important information to you. Tell me in your own words what you understood."

The advanced level is appropriate for those individuals who successfully incorporate the behavioral objectives of the survival level into their daily lives and are ready for advanced skills. They often express a desire for more information. At this stage, patients should be able to:

- Differentiate between the types of diabetes
- Recognize and remedy symptoms of uncontrolled diabetes
- Perform self-assessment, determine how well they are controlling their diabetes based on DEEDS, and make appropriate treatment decisions
- Identify the actions of oral hypoglycemic agents and/or insulin
- Adhere to a prescribed personal meal plan and anticipate and make changes as needed
- Describe the benefits and precautions of exercise, and adjust their programs as needed
- Identify and avoid over-the-counter prescription drugs that influence blood glucose
- Make appropriate treatment adjustments based on self-monitoring results (either blood, urine, or glycosylated hemoglobin)
- Follow sick-day rules including urine ketone testing
- Recognize, treat, and troubleshoot to prevent recurrence of hypoglycemia
- Freely discuss feelings about diabetes and show emotional adjustment and acceptance
- Contact health-care provider with any indication of impending problem with the feet, eyes, or gums
- Maintain good control of diabetes to prevent and delay long-term complications
- Identify available resources for special needs, such as visual impairment, weight management, traveling, or smoking cessation
- Recognize the need for ongoing diabetes education

The ongoing level of education involves continuous review and reinforcement of previously learned skills. All patients require ongoing education and maintenance of optimum diabetes care. Desirable teaching intervals depend on patients' needs and providers' preference. Well-controlled individuals can be seen every three to six months, and should be able to:[13]

- Actively participate in follow-up care by keeping scheduled appointments
- Describe and perform self-care responsibilities: "Tell me what you know about diabetes and how you are managing it at home."[14] Above all, the foundation of diabetes education is "Repeat, repeat, and repeat."

In summary, education is an integral component of diabetes treatment. Living with diabetes is a day-to-day problem. Patients need to master lifestyle changes to lead a healthy life and to break the syndromic effects of diabetes. The educational component in DEEDS is the glue that makes such changes possible. A simple reminder: "Knowledge and understanding are not just part of the treatment, they *are* the treatment."[15]

## References

1. *Diabetes in America.* US Department of Health and Human Services publication No. NIH 85-1468, Government Printing Office, 1985.

2. ADA Task Force on Financing Quality Health Care for Persons with Diabetes: *Diabetes Outpatient Education: The Evidence of Cost Savings.* Alexandria, Va, American Diabetes Association, 1986.

3. American Diabetes Association: *The Physician's Guide to Type II Diabetes (NIDDM): Diagnosis and Treatment.* Alexandria, Va, American Diabetes Association, 1984, p 13.

4. Teza S, DeVito A, Jiss R: *Resource Manual to Help Your Program Meet National Standards.* Atlanta, Ga, Centers for Disease Control, 1987.

5. Assal J, Muhlhauser I, Pernet A, et al: Patient education as the basis for diabetes care in clinical practice and research. *Diabetologia* 1985;28:602-613.

6. Falvo D: *Effective Patient Education. A Guide to Increased Compliance.* Rockville, Md, Aspen Publishers, Inc., 1985.

7. Haes W: Patient education: A component of health education. *Patient Counselling and Health Education* 1984;4:95-102.

8. Krall L: *Education, A Treatment for Diabetes*, in Krall L (ed): *Joslin's Diabetes Mellitus,* ed 11. Philadelphia, Lea & Febiger, 1985, pp 465-483.

9. Guthrie D, Guthrie R: *Nursing Management of Diabetes Mellitus.* St. Louis, CV Mosby Co, 1982.

10. Etzwiler D: Diabetes Education: How to improve practice. Princeton, *Excerpta Medica*, 1983, 277-285.

11. Bille D: *Practical Approaches to Patient Teaching.* Boston, Little Brown & Co, 1986.

12. American Association of Diabetes Educators: AADE position statement: Individualization of diabetes education and management, in *Guidelines for Diabetes Care.* New York, American Diabetes Association and the American Association of Diabetes Educators, 1982.

13. Bille D: An approach to individualizing instruction: Assessing patient readiness and activities of daily living, in AADE Module No. 1. American Association of Diabetes Educators, 1984.

14. Franz M, Krosnick A, Maschak-Carey B, et al: *Goals for Diabetes Education.* Alexandria, Va, American Diabetes Association, 1986.

15. Krall L: *Joslin Diabetes Manual.* Philadelphia, Lea & Febiger, 1978.

# Drug Therapy for Diabetes

**Alan J. Garber, MD, PhD**
**Professor of Medicine, Biochemistry,**
**and Cell Biology**
**Baylor College of Medicine**
**Chief, Diabetes/Metabolism Unit**
**The Methodist Hospital**
**Houston, TX**

Diabetes results from an absolute or relative deficiency of insulin secretion and action. In patients for whom diet and exercise fail to produce normal glycemic control, oral agents and/or insulin are essential in normalizing glucose and lipid metabolism and obviating the atherosclerotic process that accounts for up to 84 percent of all diabetic mortality.[1,2] When one to three months of diet and exercise therapy fail to produce glycemic control, pharmacologic therapy must be used to normalize glucose levels.

Oral sulfonylurea agents, particularly second-generation drugs, directly address the fundamental biochemical abnormalities of diabetes mellitus.[3] Such abnormalities result in sluggish, delayed, and inadequate insulin secretion as well as the acquired resistance to insulin action that is commonly seen in obese Type II patients. Sulfonylureas, particularly the so-called second-generation agents, augment the quantity of released insulin. They also accelerate insulin secretion to overcome the sluggish release that characterizes Type II diabetes. These effects are of basic importance in dealing with the failure of postprandial glycemic control that is often so striking in seemingly otherwise well-controlled patients with diabetes. Insulinotrophic second-generation sulfonylureas, such as glipizide, are a notable pharmacologic development.[4,5] Second-generation sulfonylureas, for the first time, allow consistent reproduction of normal glycemic control in the postprandial state. This phenomenon was never before possible in patients with diabetes mellitus. Of course, sulfonylureas can only be used for Type II disease.

In mildly to moderately obese patients, relatively severe insulin deficiency and sluggish release appear to be best managed with glipizide therapy. In such patients, this should be the therapy of choice for producing the required insulinotrophic action.

For patients with severe to morbid obesity, failures of insulin secretion are not the principal cause of diabetes mellitus.[6] Instead, massive resistance to insulin action accompanies the morbidly obese state, and additional insulin rarely solves the problem. Second-generation sulfonylureas, which are potent insulin sensitizers, greatly augment the otherwise high level of endogenous insulin and, for the first time, may provide some improvement in glycemic control.

Insulin-dependent diabetes patients and severely advanced Type II patients who have rather profound states of pancreatic islet cell exhaustion or severe insulin resistance can be managed only with insulin therapy. In the past, one or two injections of intermediate-acting insulin were considered proper therapy for these patients. Unfortunately, this form of therapy produces unphysiologic glucose regulation. In the nondiabetic state, insulin is secreted in rather large boluses that are released upon meal ingestion. Following this, relatively small quantities of insulin move slowly into circulation when the patient is not eating.

Management strategies that produce con-

Please see full Glucotrol® (glipizide) prescribing information on the last pages of this book.

sistently high levels of insulin through intermediate-acting insulin alone do not produce adequate insulinization of the patient during meals.[7,8] In fact, these strategies may produce hyperinsulinemia sufficient to accelerate the atherosclerotic process.

### Current Trends in Therapy

Current trends in treating insulin-dependent patients focus on administering increasing quantities of rapidly acting, normo-insulin prior to meals. The Type I patient may or may not require long-acting or ultra-long-acting insulin to produce adequate background levels capable of suppressing underlying ketogenesis between meals. On the other hand, with Type II patients whose disease is severe enough to require insulin therapy, such background insulinization is often not necessary or might be better administered with two small split doses of intermediate-acting insulin, such as NPH or Lente insulin. For these patients, the bulk of insulin action should be brought about by the use of rapid-acting insulin.

It is commonly believed that patients object to multiple daily injections of insulin. And while this is often true, patients who are allowed to share in making decisions about their care—those who understand that frequent regular insulin boluses produce physiologic glucose regulation and may well prevent diabetic complications and produce the greatest freedom—often become enthusiastic adherents of intensified insulin therapy.

Drug therapy for patients with diabetes increasingly focuses on attempts to recreate the normal nondiabetic pattern of glucose regulation. The newer, second-generation oral sulfonylurea agents exert their action in part by reversing the disturbed biochemical mechanisms producing Type II diabetes. These are therefore the preferred agents at the present time. Alternatively, multidose regular insulin or combination insulin/sulfonylurea therapy may be chosen if oral agents alone fail.

For the future, nonsulfonylurea oral agents are under development. These will augment and extend the efficacy of existing second-generation sulfonylureas, thereby providing their benefits to a significant number of Type II patients who would otherwise require insulin therapy.

See Patient Handouts: *Tips For Using Insulin Successfully* and *Tips For Using Sulfonylureas Successfully.*

### *References*

1. Barrett-Connor E, Orchard T: Diabetes and heart disease, in MI Harris, RE Hamman (eds): *Diabetes in America*, NIH publication No. 85-1468, 1985; pp 1-41.
2. Harris MI, Entmacher, P. Mortality from diabetes, in MI Harris, RE Hamman (eds): *Diabetes in America.* NIH publication No. 85-1468, 1985; pp 1-48.
3. Gerich JE: Sulfonylureas in the treatment of diabetes mellitus—1985. *Mayo Clin Proc* 1985;60:439-443.
4. Lebovitz HE, Feinglos MN: Mechanism of action of the second-generation sulfonylurea glipizide. *Am J Med* 1983(suppl);75:46-54.
5. Ryan JR, Jain AK, McMahon FG: Short-term glucose and insulin response to glipizide treatment in patients with non-insulin dependent diabetes mellitus, in *Glipizide: A Worldwide Clinical Review.* Princeton, *Excerpta Medica*, 1984, pp 234-247.
6. Simonson DC, Ferrannini E, Bevilacqua S, et al: Mechanism of improvement in glucose metabolism after chronic glyburide therapy. *Diabetes* 1984;33:838-845.
7. Gertler MM, Leetma HE, Saluste E, et al: Ischemic heart disease: Insulin, carbohydrate, and lipid interrelationships. *Circulation* 1972;46:103-111.
8. Wilson DE: Excessive insulin therapy: Biochemical effects and clinical repercussions. *Ann Intern Med* 1983;98:219-227.

**Patient Handouts**

## INSULIN

### Tips For Using Insulin Successfully

- Always doublecheck the insulin vial (once at the time of purchase and again before using) for proper brand, type, species, and expiration date. On first use, date the insulin vial and use it within three months to ensure stability of the insulin.
- If insulin is stored in the refrigerator, allow it to return to room temperature before injection. This will decrease the stinging sensation and the possibility of localized allergic reaction.
- Never shake the bottle. Instead, roll it gently to avoid damage to the insulin molecules.
- Always check the insulin bottle for "frosting" and for sediment that does not dissolve when the bottle is rolled.
- Insulin should be taken one half hour before meals.
- When mixing insulins, inject within five minutes to prevent alteration of action.
- When preparing the injection site, allow alcohol to dry fully. This will prevent irritation and the burning sensation.
- Never change insulin brands without consulting your physician or nurse educator.
- In case of breakage or depletion, always keep an extra bottle of each type of insulin.
- When planning to exercise shortly after injecting insulin, avoid injecting in the arms or legs. Instead, inject into the abdomen or buttocks to control absorption.
- Avoid hot showers, hot tubs, and whirlpools immediately after injecting insulin. They increase circulation and accelerate insulin action. (Avoid temperatures over 98° F or avoid completely if neuropathy is present.)
- When traveling, keep insulin in a purse or insulated container. This will prevent loss or exposure to extreme temperatures or direct sunlight.
- When traveling across time zones, ask your doctor or nurse educator to assist you in adjusting your insulin dosage.

## SULFONYLUREAS

### Tips For Using Sulfonylureas Successfully

- Establish a medication schedule and follow it as closely as possible.
- Develop the habit of routinely checking the expiration date of your medication.
- If taking a first-generation oral agent, be aware of possible reaction to alcohol ingestion.
- Check with your physician or nurse educator to determine the proper timing of medication.
- If poor memory is a problem, use a daily diary or calendar to record date and time medication is taken.
- Always check with your physician or nurse educator before changing dosage or taking additional medications, including over-the-counter drugs.

**Copy This Page for Patient Information**

# Self-Monitoring and Daily Management

**Ann Marie Cirkot, RN, BSN, CDE**
**Community Health Care Plan**
**Long Wharf Health Center**
**New Haven, CT**

The goal of diabetes treatment is the normalization of blood glucose levels, and self-monitoring is essential to achieve this goal. Patients who learn self-care responsibilities gain substantial daily control over their disease and stand a better chance of preventing long-term complications.

By definition, self-monitoring is the process of measuring blood glucose or ketone levels in urine. Its aim is to provide a useful analysis of diabetes control in relation to therapy. Only through self-monitoring can patients discharge the responsibilities that accompany diabetes mellitus.

Currently, two methods are considered effective for monitoring the daily control of diabetes. One is the urine test for glucose, an indirect measurement of blood glucose levels. The second and preferred method of self-monitoring is the direct measurement of blood glucose. In addition, a laboratory test for glycohemoglobin can aid in the assessment of glucose control.

The 1985 American Diabetes Association Policy Statement, entitled "Self-Monitoring of Blood Glucose," declared that blood glucose values are more reliable than urine test results.[1] "For this reason, self-monitoring of blood glucose (SMBG) has become an increasingly important source of clinical information, which, if used properly, will assist with diabetes control and therapy," the policy statement said.

In a 1983 position statement, the American Association of Diabetes Educators also endorsed blood glucose monitoring.[2] Abstracts presented at the 1986 Consensus Development Conference on Self-Blood Glucose Monitoring supported this method.[3]

## Urine Testing

A variety of methods are available for testing urine glucose. Although all can reliably estimate the amount of glucose in urine, they have shortcomings. One is that they are not sensitive to variations in renal threshold. As glucose levels approach 180 mg/dL, the normal renal threshold—capacity of the renal tubules to reabsorb glucose—is exceeded, and glucose is excreted into the urine.[4] The normal threshold varies among patients and may fluctuate daily in an individual patient. In older adults, the renal threshold may be as high as 240 mg/dL (high renal threshold) while the urine test remains negative.[5] On the other hand, the renal threshold is usually much lower than normal in children and during pregnancy, so that urine results in these patients will not correlate well with blood glucose.

Urine testing has another disadvantage in that for tight control it is essential to know when blood glucose is too low, but urine determinations do not supply this information. If urine testing is used as the basis for managing patients, optimum glucose control is not likely. In fact, urine testing should be reserved for patients who cannot or will not test their own blood.[6,7]

If urine testing is selected as the method of monitoring, the health-care provider should help the patient choose an appropriate product. It should also be borne in mind that certain substances can distort test results. For example, with Clinitest®, large doses of vitamin C, aspirin, and antibiotics can cause a false-positive reading. The presence of ketones can affect the result obtained with Diastix®. Tes-tape® results can be altered by moisture without visible indication. The accuracy of Chemstrip uG®, the least problematic product, is affected only by large doses of vitamin C. With all such products, accurate interpretation of results depends upon precise timing and accurate color vision.

### Urine Glucose Testing Guidelines

The time and frequency of urine glucose testing will depend on patients' current level of control and the type of treatment. Remember that a negative urine test indicates only that the blood glucose is under 180 mg/dL, and cannot indicate hypoglycemia.

The frequency of urine testing should be increased during periods of illness, bouts of hyperglycemia, and when the treatment plan is changed. To determine a meaningful pattern of results, patients must record urine glucose readings regularly. Treatment adjustments should be based only on changes in pattern, and never on an isolated reading. As an adjunct to urine testing, a broader picture of diabetes control can be derived from the glycohemoglobin test, which is described below.

### Urine Ketone Testing Guidelines

During periods of uncontrolled diabetes, urine ketone tests are used to detect the presence of acetone, an indicator of fatty-tissue breakdown. Left unchecked, acetone can accumulate in the body and precipitate diabetic ketoacidosis. Prevention of serious complications is associated with educating the patient to test for urine ketones when the blood is 240 mg/dL or higher, the urine glucose is one percent or more, and during illness or periods of severe caloric deprivation, as might occur following dental surgery. Fortunately, compared with their Type I counterparts, most individuals with Type II diabetes are not prone to ketosis.

### Glycohemoglobin Test

Glycohemoglobin, which is a relatively new laboratory measure of blood glucose control, has assumed an important role in diabetes management. Glycohemoglobin is no more than a blood glucose test with a memory. More technically, it's a time-integrated measurement of the prevailing glucose concentration to which red cells have been exposed.[6] The glycohemoglobin test can indicate effects changes in therapy have made four to eight weeks prior to the test.[7] The most important clinical use of this test is to verify self-measured blood glucose findings. This is useful because some patients inaccurately report their findings as a result of poor technique or because they feel driven to meet control expectations. Either of these situations will create a false picture of control, and may lead to flawed treatment decisions. The glycohemoglobin test will uncover the truth about control and will introduce an opportunity for an honest discussion of the treatment plan. The test also can be used as an educational tool by giving patients concrete feedback on the success of their changes.

Glycohemoglobin test results are expressed as percentages (eg, 4.5 percent to 8.5 percent) and because laboratory standards vary, interpretations must be made accordingly. In addition, the factors in Table 1 may falsely elevate or lower the result.[7]

No rules govern frequency of testing, but glycohemoglobin determinations at three- to six-month intervals allow a minimum assessment of control.

### Self-Monitoring of Blood Glucose (SMBG)

In monitoring their own blood glucose levels, patients take a drop of blood from their fingertips or earlobes and place it onto a reagent strip for a

Table 1

**Factors Affecting Glycohemoglobin Testing**

| False elevations | False low levels |
|---|---|
| Uremia | Uremia |
| Aspirin use | Anemia |
| Fetal hemoglobin | Variant hemoglobins: HbS, C, and D |
| Alcoholism | Erythropoiesis as in pregnancy |

From Rifkin,[7] p 48.

specified period of time. After that time has elapsed, the glucose value is determined by comparing the color of the reagent strip with a color chart. Alternatively, the strip may be placed in a blood glucose meter for a more precise reading. The result is then entered into a record book and interpreted in relation to an individual's treatment plan. SMBG yields information about daily variations in blood glucose and facilitates appropriate adjustments to treatment. While SMBG results alone should not be the goal of treatment, they can be invaluable in achieving treatment goals.

Advantages and disadvantages of this monitoring method must be presented to the patient, who will then be able to make an informed decision about the suitability of self-monitoring. It is generally accepted that the advantages greatly outweigh the disadvantages.[2]

### Advantages of SMBG

- More accurate than urine testing
- Practical and accepted by most patients
- Faster and cleaner than urine testing
- Results immediately available for treatment adjustment
- Confirms hypoglycemia while urine testing fails to do so
- Increases patients' understanding of diabetes
- Determines effect of exercise on blood glucose
- Assists in adherence to diet
- Helps patients gain feeling of control
- Avoids unnecessary hospitalizations during acute illness
- Helps identify factors that increase blood sugar
- Helps patients recognize symptoms of hypo- and hyperglycemia
- Improves communication between patients and providers
- Helps patients execute self-care responsibilities
- Can document blood glucose readings at home in a familiar environment

### Disadvantages of SMBG

- More expensive than urine testing
- Causes discomfort and requires time and commitment
- Flaws in technique may yield inaccurate results and lead to improper therapeutic decisions
- Technically more difficult than urine testing
- Could encourage preoccupation with SMBG results instead of treatment plan

### SMBG Candidates

The opportunity to choose SMBG should be restricted to patients who are physically and technically able, have the financial support, and are willing to make the commitment. Especially appropriate candidates include individuals who:

- Are pregnant or planning to become pregnant
- Experience frequent and/or wide, or erratic swings in blood glucose levels
- Experience severe hypoglycemic episodes
- Are insulin dependent
- Exhibit complications of the disease, ie, renal, retinal, or neural
- Are unable to maintain a regular routine
- Use insulin infusion pumps
- Have difficulty maintaining control with oral agents
- Are newly diagnosed Type II patients who have difficulty understanding the effects of food on blood glucose levels
- Are Type II patients on weight-loss programs[8]
- Are Type II patients on oral agents to attain and maintain optimum control

### Guidelines for Self-Monitoring of Blood Glucose

The time and frequency of SMBG is a direct function of the treatment goals and current state of control. Because control states and treatment goals vary, monitoring schedules must be tailor-made. As a general rule, the more intense the therapy, the more frequent the testing. The frequency should be increased during times of illness, infection, or severe stress.

With SMBG, initial blood glucose testing should be done before each meal and at bedtime. After a stable pattern is achieved, a schedule should be set to evaluate prebreakfast blood glucose levels three to seven times each week. A preprandial profile consisting of four tests should be developed one to three times a month.[6]

A determination of postprandial blood glucose excursion is helpful in portraying the meal effect and allowing for adjustments in portions and timing. Obtaining a glycohemoglobin test at three- to six-month intervals assists in determining glucose control and provides verification of SMBG results.

The American Diabetes Association has established target blood glucose levels as a general guide that providers can use in individualizing a patient's treatment goal (Table 2).[7] The health-care provider is responsible for establishing an individualized target range for blood glucose tests taken throughout the day.

An example of such individualization would be avoiding tight control with cardiac patients to reduce the risk of hypoglycemia. Also, the erratic eating habits that are common among the elderly increase their risk of hypoglycemia. Control ranges for these patients should be determined with this in mind. In addition, patients who do not experience the warning signs of hypoglycemia, either as a consequence of neuropathy or use of adrenergic-$\beta$-blockers, should not be tightly controlled. These citations illustrate the importance of tailoring the therapeutic goal individually.

Numerous products are now available for monitoring blood glucose. The choice will depend upon a patient's needs and abilities. Finances and color vision are the two factors that most often influence the choice between the meter or visual color readings. Although it is less expensive, the visual method can be interpreted only by patients who have adequate color vision. The blood glucose meter is the method of choice for Type II patients who do not have vision problems but retain the requisite technical skills. When such skills are lacking, a family member or friend may be called on to help perform testing.

Appropriate treatment decisions depend completely on accurate and reliable test results. Because many inaccurate readings are the fault of poor technique, proper and thorough patient training is essential. It has been documented that demonstration by the provider, followed by the

Table 2

**Top Limits for Glucose Control**

| | Fasting plasma glucose (mg/dL) | Two-hour postprandial plasma glucose (mg/dL) |
|---|---|---|
| Normal | 115 | 140 |
| Acceptable | 140 | 200 |
| Poor | >200 | >235 |

Note: These values reflect tight control and may not apply universally. For elderly patients, increase values by approximately 10%.

From Rifkin,[7] p 25.

patient's repetition of the activity, is the best way to improve learning self-care skills. It is also true that an often neglected aspect of SMBG is adequate follow-up. Periodic assessment of self-monitoring techniques can help ensure reliable results and also reveal vision problems that preclude continuation of the visual method.

Merely obtaining and documenting SMBG results is not enough. Instead, the health-care professional must give each patient clear written instructions that specify what must be done with the results. Blood glucose goals must be clearly stated and changes in treatment must be identified carefully. Without such direction, the SMBG records are virtually useless. (See Table 3, Self-Monitoring Record.)

While self-monitoring is only one component of diabetes management, it is the best tool available for making treatment changes that lead to diabetes control. The choice of method for self-monitoring, the schedule for testing, and the treatment goal itself must be carefully tailored to each patient. Ongoing patient education is essential to this process.

### Short-Term Problems: Hypoglycemia and Hyperglycemia

In 1982, Guthrie and Guthrie stated that one of the most alarming and least understood areas of diabetes treatment is distinguishing between hypoglycemia and hyperglycemia.[4] Treating these conditions is no less problematic. Managing these acute complications depends on a thorough knowledge of associated concepts. Once clinicians have learned to recognize and treat these problems, they will find it easier than ever to educate patients in causes, prevention, and treatment.

### Hypoglycemia

Hypoglycemia, which is commonly described as an insulin reaction or "low blood sugar," occurs when a drop in the blood glucose level stimulates a sequence of physiological events. Generally, blood glucose levels of 50 mg/dL or less are considered hypoglycemic.[7] However, this value must be adjusted according to individual circumstances. This discussion centers on patients whose management depends on pharmaceutical therapy.

Table 3

## Self-Monitoring Record for Lifestyle Changes

NAME: ______________________________ PHONE: ________________

ADDRESS: ____________________________________________

____________________________________________________

Physician: ____________________________ Phone: ________________

Educator: ____________________________ Phone: ________________

### Diabetes Treatment Guide

My treatment goal is ______ blood glucose before meals.
______ blood glucose after meals.

If my blood glucose is below ______ or above ______, I must contact my physician.

If my blood glucose is 240 or more I should test my urine for ketones. If positive for ketones, I must contact my physician.

I must treat all symptoms of low blood sugar (____ or less) immediately and contact my physician.

Signs & Symptoms:
- sweatiness
- dizziness
- shakiness
- nervousness
- weakness
- irritability
- headache
- excessive dreaming

Treatment: 4 oz regular soda or juice or
2 teaspoons of honey or syrup or
Commercial glucose product
Repeat treatment if needed
Always follow treatment with a snack.

### Blood Glucose Test Record

| DATE | MED | Breakfast Test | | MED | Lunch Test | | MED | Dinner Test | | OTHER | COMMENTS |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | before | after | | before | after | | before | after | | |
| | | | | | | | | | | | |
| | | | | | | | | | | | |
| | | | | | | | | | | | |

Hypoglycemia is most common among Type I patients. Such patients who have never experienced a hypoglycemic episode need to have their control status evaluated. Individuals with Type II diabetes rarely encounter severe hypoglycemia. Even so, those who take oral agents can experience prolonged episodes that last for days because of the long half-lives of some drugs. In addition, renal and liver disease can produce a buildup of drug levels which may result in low blood glucose.

Many individuals with diabetes experience hypoglycemia without undue complications. Yet, it is well known that, unrecognized and untreated, this condition can cause coma and, on rare occasions, even death. Strictly controlled patients are often on the border between acceptable blood glucose levels and low ones. No more than a slight scheduling delay in a meal or snack can easily lower their glucose levels and produce serious problems in such everyday activities as driving a car. Fortunately, self-monitoring has greatly enhanced our ability to prevent hypoglycemia. This is possible only if patients are taught strategies for preventing this short-term problem.

Hypoglycemia is caused by too much medication, not enough food intake, increased activity without adequate food intake, and/or alcohol ingestion, which interferes with the release of glycogen from the liver. In planning for prevention, it is essential to determine the exact cause.

The signs and symptoms of hypoglycemia occur in two stages and depend on how quickly and to what level the blood glucose falls. In the adrenal stage (which is the response to stress), the release of epinephrine causes sweating, hunger, tremor and tachycardia, palpitations, anxiety and nervousness, irritability, and, occasionally, nausea and vomiting.[9] These are "warning signs" that announce the need for immediate treatment. Some individuals, especially patients taking $\beta$-blockers, may not experience these symptoms. This may also be true of patients who have had diabetes for many years and whose symptoms are masked. Many elderly patients fall into this category and experience hypoglycemia without knowing it. Another confusing factor is that symptoms of hypoglycemia can be mistaken for stroke in older patients. Excessive caffeine intake can also mimic hypoglycemia's symptoms.

When hypoglycemia goes untreated, blood glucose continues to fall and advances to the cerebral stage, which is characterized by symptoms that result from the decreased availability of glucose to the brain. The transition from the adrenal stage to the cerebral stage can occur within as few as three to six minutes.[9] In general, the progression is from muscular incoordination and cerebral inadequacy to a semicomatose state, and then to convulsions and complete unconsciousness. Cerebral stage symptoms include:[9]

- Headache and visual disturbance
- Confusion
- Garbled or slurred speech
- Unsteady gait
- Tingling of the mouth and fingers
- Personality changes, including belligerence, depression or psychotic behavior
- Semicoma with muscular twitching and convulsions
- Complete unconsciousness

Signs and symptoms of hypoglycemia vary among patients. Individuals may experience some, all, or none of these. To determine whether or not a patient has experienced hypoglycemia, ask whether he has ever broken out in a cold sweat or felt weak, shaky, dizzy, extremely hungry prior to eating, or felt "hung over" in the morning. Nocturnal hypoglycemia can be signaled by restless sleep, excessive sweating, headache, nightmares, rolling and moaning, and even falling out of bed. The spouse is often the first to recognize these signs and should be made aware of the clues. Because hypoglycemia can easily be mistaken for intoxication, treatment is sometimes delayed. Consequently, identification bracelets or necklaces are mandatory for all patients on diabetes drug therapy.

The immediate intake of some form of fast-acting glucose to reverse the falling blood glucose is the treatment for hypoglycemia. For conscious patients, therapy should be 15 g of a simple carbohydrate, as can be derived from one of the following:[10]

- ½ cup orange or apple juice
- 1 tablespoon honey or syrup
- ½ cup cola (not sugar-free)
- 2 tablespoons raisins

Nonfood sources include B-D Glucose Tablets®and Monoject Insulin Reaction Gel® . Remember that liquids are preferred because they are absorbed more quickly. Chocolate bars, with their high-fat content retard absorption of glucose and are not recommended.

If possible, blood glucose should be measured during a hypoglycemic episode without delaying treatment unduly. A blood glucose test is the only way to confirm hypoglycemia. Testing glucose levels 15 minutes after treatment indicates the degree of improvement and determines the need to repeat the treatment. Urine testing has no value during hypoglycemic episodes because negative results can occur in too wide a blood glucose range.

Treatment of unconscious patients requires the injection of glucagon, a hormone that raises the blood glucose level by stimulating glycogenolysis. This prescription drug is injected like insulin and requires careful education of the patient's family for proper administration. Most often, this drug is recommended for patients on insulin therapy.

After initial treatment for hypoglycemia (unless it is mealtime), an extra snack should be eaten to prevent recurrence. Many patients are embarrassed after experiencing hypoglycemia and need support and understanding, not blame and accusation. Overtreatment is the most common post-hypoglycemic problem and can be avoided by patient education.

The best therapeutic response to hypoglycemia is prevention, and time is well spent educating patients and their families in methods of accomplishing this. The need to follow the dietary regimen and not to skip meals or snacks should be emphasized, especially with older patients. Counselors must be certain that diabetics understand their drug therapy, and they must teach patients not to take double doses of medication if a scheduled dose is missed, unless authorized by the provider.

Patients also should be taught to report all insulin reactions to a health-care provider to discuss the cause and develop a prevention plan. Patients also need to be instructed to keep a record of treatments they have used so that a personalized plan can be developed. Another practical tip is to carry some form of fast-acting glucose that can help them prepare for an unexpected meal delay; choosing a commercial product instead of candy will reduce the temptation to snack. If the patient anticipates increased activity, a delayed meal, or alcohol consumption, he should know how to compensate with dietary adjustments.

Patients who live alone should develop a safety system in which a relative or neighbor makes daily contact to verify their condition. Finally, self-monitoring of blood glucose is an invaluable tool that will provide prevention and early detection of low blood glucose as well as confirm existing hypoglycemia.

### Hyperglycemia

Hyperglycemia, also known as high blood glucose, is "diabetes out of control." It occurs when fasting blood glucose values rise above 180 mg/dL. Short-term hyperglycemia usually lasts less than two days and does not produce more serious complications. However, prolonged and untreated hyperglycemia can result in serious long-term complications.

Most commonly, the gradual onset of hyperglycemia is a function of insufficient medication. Other causes include diminished activity, infection, and excessive food intake. Severe emotional or physical stress, such as might be caused by the death of a loved one, surgery, or major illness, can also elevate blood glucose. Certain drugs (corticosteroids, thiazides, Dilantin® , sugared cough syrups, oral contraceptives, and anti-inflammatory agents) may precipitate hyperglycemia. As is true of hypoglycemia, education is the key to prevention.

The three classic signs and symptoms of hyperglycemia are polyuria, polydipsia, and polyphagia. Other signs and symptoms include dry mouth ("cotton mouth"), blurred vision, fatigue, dry and itchy skin, frequent skin and vaginal infections, and occasionally, weight loss. Symptoms of ketoacidosis include nausea, vomiting, abdominal pain, Kussmaul respiration, acetone breath, and dehydration. Untreated, this condition can lead to coma and death. All who participate in managing patients with diabetes should know that hyperglycemia is not always accompanied by these signs and symptoms. Individuals may experience some, all, or none of the warning signs. This fact demonstrates the importance of self-monitoring to detect rises in blood glucose.

Effective treatment of hyperglycemia requires identification of its cause, followed by appropriate alterations in management. Self-monitoring is the vehicle for identifying early changes in the glucose pattern so that prolonged hyperglycemia can be prevented. If the cause is diet related, appropriate changes should be recommended. If the cause is decreased activity, adjustments in diet and medication can compensate. If insufficient medication is the culprit, the problem is easily corrected. When hyperglycemia is related to severe stress, temporary insulin (human) may be the treatment of choice for patients on diet or oral agent regimens.

### "Sick-Day" Management

"Sick-day" guidelines apply to those brief, one- to two-day periods in which diabetes patients experience acute minor illnesses, including colds, flu symptoms, or even upset stomachs. Because the associated infections increase insulin requirements, such illnesses often cause hyperglycemia. Consequently, immediate recognition and treatment are essential for preventing more serious complications. Simply stated, sick-day guidelines should help patients avoid ketosis and prevent unnecessary hospitalization.

### Guidelines for Preventing Hyperglycemia

Patients on insulin therapy must never fail to take their insulin, even if oral intake is not tolerated. In fact, at such times additional insulin may be required to control blood glucose. Individuals who use oral hypoglycemic agents should not interrupt their therapy unless persistent vomiting occurs. Temporary therapy with insulin (human) may be indicated if an illness becomes prolonged or severe. Diabetes patients who are ill must monitor their blood glucose or urine every four hours, or more often as needed. Particularly for the Type I patient, blood glucose levels of 240 mg/dL or above announce the need for a urine ketone test. The patient must be instructed to notify the health-care provider when urine ketones are present so that ap-

Table 4

**"Sick-Day" Foods**
**Based on ADA 1986 Food Exchange Lists**

If regular foods cannot be tolerated, 50 g of the following carbohydrates should be consumed every three to four hours.[11]

| Serving Size | Food | Grams (g) of Carbohydrates |
|---|---|---|
| ½ cup | Apple juice | 15 |
| ⅓ cup | Cranberry juice | 15 |
| ½ cup | Orange juice | 15 |
| ½ cup | Regular ginger ale | 10 |
| ½ cup | Regular 7-Up | 12 |
| 1 twin pop | Popsicle | 24 |
| ½ cup | Regular gelatin | 20 |
| ⅓ cup | Grape juice | 15 |
| ½ cup | Cooked cereal | 15 |
| 1 cup | Chicken noodle soup | 10 |
| ½ cup | Ice cream (vanilla) | 15 |
| ½ cup | Sherbet | 30 |
| ½ cup | Instant pudding | 30 |
| ½ cup | Custard | 15 |
| ½ cup | Fruit-flavored yogurt | 20 |
| 6 | Saltine crackers | 15 |

**Copy This Page for Patient Information**

propriate treatment can be obtained.

During illnesses, the patient's fluid and carbohydrate intake must be sufficient to prevent dehydration and catabolism. If solid foods are not tolerated, liquid meals can be substituted. The liquid meal should be divided into at least two smaller meals, preferably six, to provide a steady stream of glucose into the circulating blood. There is an exception here to the general rule of avoiding concentrated sweets. Depleted stores of potassium and sodium caused by vomiting and diarrhea must be replaced to prevent electrolyte imbalance. Specific written instructions with a sample menu are a useful part of patient education (see Table 4).

In summary, sick-day management begins with educating patients in methods of preventing potentially serious diabetic complications. Part of this process involves teaching them to recognize when such symptoms as fever, vomiting and diarrhea, and altered mental status warrant calling a physician. A specific, written plan helps the patient safely manage periods of short-term illness.

References

1. American Diabetes Association: Policy statement: Self-monitoring of blood glucose. *Diabetes Care* 1985;8:515.
2. American Association of Diabetes Educators: Position statement: Blood glucose monitoring. *The Diabetes Educator* 1983;3.
3. Centers for Disease Control, Food and Drug Administration, and National Institutes of Health (Cosponsors); (1986). *Consensus Development Conference on Self-Blood Glucose Monitoring* (November 17-19). Arlington, Va, American Diabetes Association.
4. Guthrie D, Guthrie R: *Nursing Management of Diabetes Mellitus*, ed 2. St. Louis, CV Mosby Co, 1982, pp 22, 189.
5. Hollerorth H: *Diabetes Teaching Guide*, revised ed. Boston, Ma, Joslin Diabetes Center, 1986, p 74.
6. Davidson M: *Diabetes Mellitus Diagnosis and Treatment*. New York, Wiley Medical Publications, 1986, p 298.
7. Rifkin H (ed): *The Physician's Guide to Non-Insulin Dependent (Type II) Diabetes: Diagnosis and Treatment*, ed 2. Alexandria, Va, American Diabetes Association, 1988, pp 25, 48, 78.
8. Kilo C, Dudley J: *Self Blood Glucose Monitoring for the Person with Diabetes Mellitus: A Guide for the Physician in Private Practice*. Kilo Diabetes and Vascular Research Foundation, 1984, p 19.
9. Olson OC: *Diagnosis and Management of Diabetes Mellitus*, ed 2. New York, Raven Press, 1987, pp 100, 101.
10. Kulkarni KD: Altering the basic meal plan, in *Handbook of Diabetes Nutritional Management*. Rockville, Md, Aspen Publishers, Inc, 1987, pp 174-190.
11. Lind D, Bonar J, Stone P: Managing well on sick days. *Diabetes Forecast*, February 1980, pp 22-25.

# Foot Care

**Ann Marie Cirkot, RN, BSN, CDE**
**Community Health Care Plan**
**Long Wharf Health Center**
**New Haven, CT**
**and**
**Jane Darling, RN, ANP, CDE**
**Diabetes Education Coordinator**
**Mount Sinai Hospital**
**Hartford, CT**

Individuals with diabetes are particularly vulnerable to foot problems.[1] In fact, more than half of the nontraumatic amputations performed in the United States are performed with diabetes patients, and it has been estimated that more than 50 percent of these could have been prevented with proper care.[2] Consequently, special attention must be paid to prevention and to the immediate treatment of potentially serious problems of the foot.

Problems particular to the diabetic foot are the result of neurologic and vascular disease, exogenous infection, or, more commonly, a combination of these complications. Lesions are usually found in feet that are insensitive, deformed, or ischemic. Such feet are vulnerable to trauma, which may lead to ulceration, infection, and gangrene. The lack of pain associated with diabetic foot lesions often prevents early detection of such problems, and is actually responsible for most foot problems diabetics experience. Because mild nerve-conduction defects can be reversible if treated early, emphasis should be placed on achieving optimum glucose control.[2] Table 1 outlines the warning signs and symptoms of diabetic foot problems.

## Foot-Care Principles

Health-care providers bear responsibility for educating patients about the principles of proper foot care. This is not a difficult burden, for daily care is simple, requiring no more than that the patient wash, wipe, and examine the feet with care.

*Washing:* To remove potentially dangerous bacteria, the feet should be washed every day with mild soap and water, and thoroughly rinsed. Soaking should be avoided because of the risk of infection from maceration. Avoid using such agents as Epsom salts, boric acid, or lysol. To prevent scalding, the water temperature must be checked with the back of the hand.

*Wiping:* The feet should be dried with a soft towel, and attention must be paid to the web spaces between the toes. Brisk, harsh drying can produce trauma. Applying a nonalcohol-based lotion is recommended before bedtime, but it should not be applied between the toes. For moist skin problems, talcum can be helpful.

*Inspection:* Daily inspection of the feet is the only way to identify problems at an early stage. The diagram of pressure points in the figure may be helpful. Every diabetes patient should be taught to inspect the feet for:

- Redness, swelling, discharge (infection)
- Dryness or cracking
- Corns, calluses, blisters, or sores
- Thickened nails
- Cuts and bruises
- Pain or absence of sensation
- Change in skin color or temperature
- Change in shape
- Loss of hair

Table 1

**Warning Symptoms and Signs of Diabetic Foot Problems**

| | SYMPTOMS | SIGNS |
|---|---|---|
| **Vascular** | Cold feet<br>Intermittent claudication involving calf or foot<br>Pain at rest, especially nocturnal, relieved by dependency | Absent pedal, popliteal, or femoral pulses<br>Femoral bruits<br>Dependent rubor, plantar pallor on elevation<br>Prolonged capillary filling time (>3-4 seconds)<br>Decreased skin temperature |
| **Neurologic** | Sensory: Burning, tingling, or crawling sensations, pain and hypersensitivity<br>Motor: Weakness (drop foot)<br>Autonomic: Diminished sweating | Sensory: Deficits (vibratory and proprioceptive, then pain and temperature perception), hyperesthesia<br>Motor: Diminished to absent deep tendon reflexes (Achilles then patellar), weakness<br>Autonomic: Diminished to absent sweating |
| **Musculoskeletal** | Gradual change in foot shape<br>Sudden, painless change in foot shape, with swelling, without history of trauma | Cavus feet with claw toes<br>Drop foot<br>"Rocker-bottom" foot (Charcot's joint)<br>Neuropathic arthropathy |
| **Dermatologic** | Exquisitely painful or painless wounds<br>Slow-healing or nonhealing wounds, or necrosis<br>Skin color changes (cyanosis, redness)<br>Chronic scaling, itching, or dry feet<br>Recurrent infections (paronychia, athlete's foot, etc.) | Skin<br>Abnormal dryness<br>Chronic tinea infections<br>Keratotic lesions with or without hemorrhage (plantar or digital)<br>Trophic ulcer<br>Hair<br>Diminished to absent<br>Nails<br>Trophic changes<br>Onychomycosis<br>Subungual ulceration or abscess<br>Ingrown nails with paronychia |

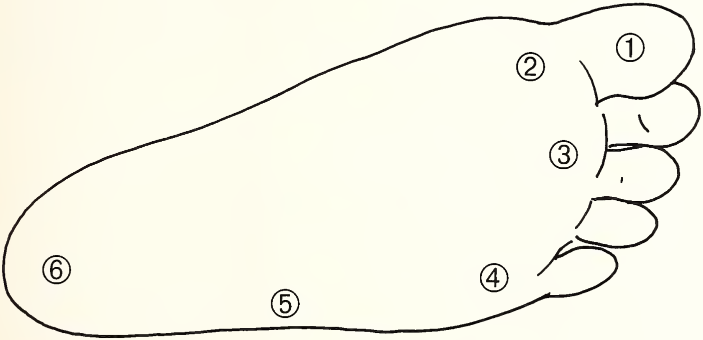
*Scardina RJ: Diabetic foot problems: Assessment and prevention.* Clinical Diabetes *1983;1(March/April):1-7.*

**Copy This Page for Patient Information**

Figure 1

## Diagram of Pressure Points

These pressure points on the bottom of the foot should be carefully inspected each day. In this way, problems can be identified at an early stage.



**Copy This Page for Patient Information**

- Peeling skin between the toes
- Unusual odor
- Absence of perspiration
- Ingrown toenails
- Signs of trauma or injury
- Loss of pedal pulses

Special attention should be given to corns and calluses. In response to pressure points and friction (Figure 1), the body naturally builds layers of protective skin. However, the cause must be identified and corrected. Referral to a podiatrist may be necessary for the removal of corns, calluses, and problem nails. Use of commercial corn-removal medicines or plasters must be avoided. Often, poorly fitting shoes are the primary cause of corns and calluses. When underlying infection is present, it must be eliminated without delay. Patients should be cautioned against performing "surgical" procedures on their own feet.

To prevent unnecessary injury, nails should not be cut, but filed straight across. Knives or razor blades should never be used. Nails that are very thick and those that are ingrown or injured require special care and should not be self-treated. Scalding can be prevented by carefully testing the water temperature and never using heating pads, hot water bottles, or heating lamps. Cold packs must also be avoided. Most important, instruct patients never to go barefoot, even at home. Emphasize that new shoes should be broken in slowly, and that it is essential to wear sturdy shoes that fit properly. Clean, color-fast socks or stockings (without holes) that do not bind should be worn and changed daily. Smoking, which greatly reduces peripheral circulation, must be discouraged.

Physical impairment or decreased acuity of vision mandates that the sense of touch be put to greater use in identifying foot problems. A mirror placed on the floor can help the physically impaired patient inspect the feet. If self-inspection is impossible, a family member must be taught to assist. The booklet *Footcare: Taking Charge of Your Diabetes* contains a patient-oriented approach to the principles of foot management.

In summary, individuals with diabetes are particularly susceptible to foot problems. Prevention of these problems occurs through patient education on the principles of foot care. The practice of daily foot care can have a significant impact on the incidence of amputation.

## References

1. Krall L: Education, a treatment for diabetes, in Krall L (ed): *Joslin's Diabetes Mellitus*, ed 11. Philadelphia, Lea & Febiger, 1985, pp 465-483.

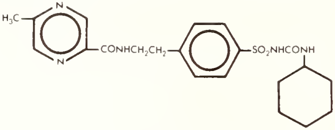
2. Rifkin H (ed): *The Physician's Guide to Type II Diabetes (NIDDM): Diagnosis and Treatment*, ed 2. Alexandria, Va, American Diabetes Association, 1988.

# GLUCOTROL®
## glipizide
## TABLETS
### *For Oral Use*

### DESCRIPTION

GLUCOTROL (glipizide) is an oral blood-glucose-lowering drug of the sulfonylurea class.

The Chemical Abstracts name of glipizide is 1-cyclohexyl-3-[[p-[2-(5-methylpyrazinecarboxamido)ethyl]phenyl]sulfonyl]urea. The molecular formula is $C_{21}H_{27}N_5O_4S$; the molecular weight is 445.55; the structural formula is shown below:



Glipizide is a whitish, odorless powder with a pKa of 5.9. It is insoluble in water and alcohols, but soluble in 0.1 *N* NaOH; it is freely soluble in dimethylformamide. GLUCOTROL tablets for oral use are available in 5 and 10 mg strengths.

Inert ingredients are: colloidal silicon dioxide; lactose; microcrystalline cellulose; starch; stearic acid.

### CLINICAL PHARMACOLOGY

**Mechanism of Action:** The primary mode of action of GLUCOTROL in experimental animals appears to be the stimulation of insulin secretion from the beta cells of pancreatic islet tissue and is thus dependent on functioning beta cells in the pancreatic islets. In humans GLUCOTROL appears to lower the blood glucose acutely by stimulating the release of insulin from the pancreas, an effect dependent upon functioning beta cells in the pancreatic islets. The mechanism by which GLUCOTROL lowers blood glucose during long-term administration has not been clearly established. In man, stimulation of insulin secretion by GLUCOTROL in response to a meal is undoubtedly of major importance. Fasting insulin levels are not elevated even on long-term GLUCOTROL administration, but the postprandial insulin response continues to be enhanced after at least 6 months of treatment. The insulinotropic response to a meal occurs within 30 minutes after an oral dose of GLUCOTROL in diabetic patients, but elevated insulin levels do not persist beyond the time of the meal challenge. Extrapancreatic effects may play a part in the mechanism of action of oral sulfonylurea hypoglycemic drugs.

Blood sugar control persists in some patients for up to 24 hours after a single dose of GLUCOTROL, even though plasma levels have declined to a small fraction of peak levels by that time (see Pharmacokinetics below).

Some patients fail to respond initially, or gradually lose their responsiveness to sulfonylurea drugs, including GLUCOTROL. Alternatively, GLUCOTROL may be effective in some patients who have not responded or have ceased to respond to other sulfonylureas.

**Other Effects:** It has been shown that GLUCOTROL therapy was effective in controlling blood sugar without deleterious changes in the plasma lipoprotein profiles of patients treated for NIDDM.

In a placebo-controlled, crossover study in normal volunteers, GLUCOTROL had no anti-diuretic activity, and, in fact, led to a slight increase in free water clearance.

**Pharmacokinetics:** Gastrointestinal absorption of GLUCOTROL in man is uniform, rapid, and essentially complete. Peak plasma concentrations occur 1-3 hours after a single oral dose. The half-life of elimination ranges from 2-4 hours in normal subjects, whether given intravenously or orally. The metabolic and excretory patterns are similar with the two routes of administration, indicating that first-pass metabolism is not significant. GLUCOTROL does not accumulate in plasma on repeated oral administration. Total absorption and disposition of an oral dose was unaffected by food in normal volunteers, but absorption was delayed by about 40 minutes. Thus GLUCOTROL was more effective when administered about 30 minutes before, rather than with, a test meal in diabetic patients. Protein binding was studied in serum from volunteers who received either oral or intravenous GLUCOTROL and found to be 98-99% one hour after either route of administration. The apparent volume of distribution of GLUCOTROL after intravenous administration was 11 liters, indicative of localization within the extracellular fluid compartment. In mice no GLUCOTROL or metabolites were detectable autoradiographically in the brain or spinal cord of males or females, nor in the fetuses of pregnant females. In another study, however, very small amounts of radioactivity were detected in the fetuses of rats given labelled drug.

The metabolism of GLUCOTROL is extensive and occurs mainly in the liver. The primary metabolites are inactive hydroxylation products and polar conjugates and are excreted mainly in the urine. Less than 10% unchanged GLUCOTROL is found in the urine.

### INDICATIONS AND USAGE

GLUCOTROL is indicated as an adjunct to diet for the control of hyperglycemia and its associated symptomatology in patients with non-insulin-dependent diabetes mellitus (NIDDM; type II), formerly known as maturity-onset diabetes, after an adequate trial of dietary therapy has proved unsatisfactory.

## INDICATIONS AND USAGE *(continued)*

In initiating treatment for non-insulin-dependent diabetes, diet should be emphasized as the primary form of treatment. Caloric restriction and weight loss are essential in the obese diabetic patient. Proper dietary management alone may be effective in controlling the blood glucose and symptoms of hyperglycemia. The importance of regular physical activity should also be stressed, and cardiovascular risk factors should be identified, and corrective measures taken where possible.

If this treatment program fails to reduce symptoms and/or blood glucose, the use of an oral sulfonylurea or insulin should be considered. Use of GLUCOTROL must be viewed by both the physician and patient as a treatment in addition to diet, and not as a substitute for diet or as a convenient mechanism for avoiding dietary restraint. Furthermore, loss of blood glucose control on diet alone also may be transient, thus requiring only short-term administration of GLUCOTROL.

During maintenance programs, GLUCOTROL should be discontinued if satisfactory lowering of blood glucose is no longer achieved. Judgments should be based on regular clinical and laboratory evaluations.

In considering the use of GLUCOTROL in asymptomatic patients, it should be recognized that controlling the blood glucose in non-insulin-dependent diabetes has not been definitely established to be effective in preventing the long-term cardiovascular or neural complications of diabetes.

## CONTRAINDICATIONS

GLUCOTROL is contraindicated in patients with:

1. Known hypersensitivity to the drug.
2. Diabetic ketoacidosis, with or without coma. This condition should be treated with insulin.

## WARNINGS

**SPECIAL WARNING ON INCREASED RISK OF CARDIOVASCULAR MORTALITY: The administration of oral hypoglycemic drugs has been reported to be associated with increased cardiovascular mortality as compared to treatment with diet alone or diet plus insulin. This warning is based on the study conducted by the University Group Diabetes Program (UGDP), a long-term prospective clinical trial designed to evaluate the effectiveness of glucose-lowering drugs in preventing or delaying vascular complications in patients with non-insulin-dependent diabetes. The study involved 823 patients who were randomly assigned to one of four treatment groups (*Diabetes*, 19, supp. 2: 747-830, 1970).**

**UGDP reported that patients treated for 5 to 8 years with diet plus a fixed dose of tolbutamide (1.5 grams per day) had a rate of cardiovascular mortality approximately 2-1/2 times that of patients treated with diet alone. A significant increase in total mortality was not observed, but the use of tolbutamide was discontinued based on the increase in cardiovascular mortality, thus limiting the opportunity for the study to show an increase in overall mortality. Despite controversy regarding the interpretation of these results, the findings of the UGDP study provide an adequate basis for this warning. The patient should be informed of the potential risks and advantages of GLUCOTROL and of alternative modes of therapy.**

**Although only one drug in the sulfonylurea class (tolbutamide) was included in this study, it is prudent from a safety standpoint to consider that this warning may also apply to other oral hypoglycemic drugs in this class, in view of their close similarities in mode of action and chemical structure.**

## PRECAUTIONS

### General

**Renal and Hepatic Disease:** The metabolism and excretion of GLUCOTROL may be slowed in patients with impaired renal and/or hepatic function. If hypoglycemia should occur in such patients, it may be prolonged and appropriate management should be instituted.

**Hypoglycemia:** All sulfonylurea drugs are capable of producing severe hypoglycemia. Proper patient selection, dosage, and instructions are important to avoid hypoglycemic episodes. Renal or hepatic insufficiency may cause elevated blood levels of GLUCOTROL and the latter may also diminish gluconeogenic capacity, both of which increase the risk of serious hypoglycemic reactions. Elderly, debilitated or malnourished patients, and those with adrenal or pituitary insufficiency are particularly susceptible to the hypoglycemic action of glucose-lowering drugs. Hypoglycemia may be difficult to recognize in the elderly, and in people who are taking beta-adrenergic blocking drugs. Hypoglycemia is more likely to occur when caloric intake is deficient, after severe or prolonged exercise, when alcohol is ingested, or when more than one glucose-lowering drug is used.

**Loss of Control of Blood Glucose:** When a patient stabilized on any diabetic regimen is exposed to stress such as fever, trauma, infection, or surgery, a loss of control may occur. At such times, it may be necessary to discontinue GLUCOTROL and administer insulin.

The effectiveness of any oral hypoglycemic drug, including GLUCOTROL, in lowering blood glucose to a desired level decreases in many patients over a period of time, which may be due to progression of the severity of the diabetes or to diminished responsiveness to the drug. This phenomenon is known as secondary failure, to distinguish it from primary failure in which the drug is ineffective in an individual patient when first given.

**Laboratory Tests:** Blood and urine glucose should be monitored periodically. Measurement of glycosylated hemoglobin may be useful.

**Information for Patients:** Patients should be informed of the potential risks and advantages of GLUCOTROL and of alternative modes of therapy. They should also be informed about the importance of adhering to dietary instructions, of a regular exercise program, and of regular testing of urine and/or blood glucose.

The risks of hypoglycemia, its symptoms and treatment, and conditions that predispose to its development should be explained to patients and responsible family members. Primary and secondary failure should also be explained.

**Drug Interactions:** The hypoglycemic action of sulfonylureas may be potentiated by certain drugs including nonsteroidal anti-inflammatory agents and other drugs that are highly protein bound, salicylates, sulfonamides, chloramphenicol, probenecid, coumarins, monoamine oxidase inhibitors, and beta adrenergic blocking agents. When such drugs are administered to a patient receiving GLUCOTROL, the patient should be observed closely for hypoglycemia. When such drugs are withdrawn from a patient receiving GLUCOTROL, the patient should be observed closely for loss of control.

## PRECAUTIONS *(continued)*

*In vitro* binding studies with human serum proteins indicate that GLUCOTROL binds differently than tolbutamide and does not interact with salicylate or dicumarol. However, caution must be exercised in extrapolating these findings to the clinical situation and in the use of GLUCOTROL with these drugs.

Certain drugs tend to produce hyperglycemia and may lead to loss of control. These drugs include the thiazides and other diuretics, corticosteroids, phenothiazines, thyroid products, estrogens, oral contraceptives, phenytoin, nicotinic acid, sympathomimetics, calcium channel blocking drugs, and isoniazid. When such drugs are administered to a patient receiving GLUCOTROL, the patient should be closely observed for loss of control. When such drugs are withdrawn from a patient receiving GLUCOTROL, the patient should be observed closely for hypoglycemia.

A potential interaction between oral miconazole and oral hypoglycemic agents leading to severe hypoglycemia has been reported. Whether this interaction also occurs with the intravenous, topical, or vaginal preparations of miconazole is not known.

**Carcinogenesis, Mutagenesis, Impairment of Fertility:** A twenty month study in rats and an eighteen month study in mice at doses up to 75 times the maximum human dose revealed no evidence of drug-related carcinogenicity. Bacterial and *in vivo* mutagenicity tests were uniformly negative. Studies in rats of both sexes at doses up to 75 times the human dose showed no effects on fertility.

**Pregnancy:** Pregnancy Category C: GLUCOTROL (glipizide) was found to be mildly fetotoxic in rat reproductive studies at all dose levels (5-50 mg/kg). This fetotoxicity has been similarly noted with other sulfonylureas, such as tolbutamide and tolazamide. The effect is perinatal and believed to be directly related to the pharmacologic (hypoglycemic) action of GLUCOTROL. In studies in rats and rabbits no teratogenic effects were found. There are no adequate and well controlled studies in pregnant women. GLUCOTROL should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Because recent information suggests that abnormal blood glucose levels during pregnancy are associated with a higher incidence of congenital abnormalities, many experts recommend that insulin be used during pregnancy to maintain blood glucose levels as close to normal as possible.

**Nonteratogenic Effects:** Prolonged severe hypoglycemia (4 to 10 days) has been reported in neonates born to mothers who were receiving a sulfonylurea drug at the time of delivery. This has been reported more frequently with the use of agents with prolonged half-lives. If GLUCOTROL is used during pregnancy, it should be discontinued at least one month before the expected delivery date.

**Nursing Mothers:** Although it is not known whether GLUCOTROL is excreted in human milk, some sulfonylurea drugs are known to be excreted in human milk. Because the potential for hypoglycemia in nursing infants may exist, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. If the drug is discontinued and if diet alone is inadequate for controlling blood glucose, insulin therapy should be considered.

**Pediatric Use:** Safety and effectiveness in children have not been established.

## ADVERSE REACTIONS

In U.S. and foreign controlled studies, the frequency of serious adverse reactions reported was very low. Of 702 patients, 11.8% reported adverse reactions and in only 1.5% was GLUCOTROL discontinued.

**Hypoglycemia:** See PRECAUTIONS and OVERDOSAGE sections.

**Gastrointestinal:** Gastrointestinal disturbances are the most common reactions. Gastrointestinal complaints were reported with the following approximate incidence: nausea and diarrhea, one in seventy; constipation and gastralgia, one in one hundred. They appear to be dose-related and may disappear on division or reduction of dosage. Cholestatic jaundice may occur rarely with sulfonylureas: GLUCOTROL should be discontinued if this occurs.

**Dermatologic:** Allergic skin reactions including erythema, morbilliform or maculopapular eruptions, urticaria, pruritus, and eczema have been reported in about one in seventy patients. These may be transient and may disappear despite continued use of GLUCOTROL; if skin reactions persist, the drug should be discontinued. Porphyria cutanea tarda and photosensitivity reactions have been reported with sulfonylureas.

**Hematologic:** Leukopenia, agranulocytosis, thrombocytopenia, hemolytic anemia, aplastic anemia, and pancytopenia have been reported with sulfonylureas.

**Metabolic:** Hepatic porphyria and disulfiram-like reactions have been reported with sulfonylureas. In the mouse, GLUCOTROL pretreatment did not cause an accumulation of acetaldehyde after ethanol administration. Clinical experience to date has shown that GLUCOTROL has an extremely low incidence of disulfiram-like alcohol reactions.

**Endocrine Reactions:** Cases of hyponatremia and the syndrome of inappropriate antidiuretic hormone (SIADH) secretion have been reported with this and other sulfonylureas.

**Miscellaneous:** Dizziness, drowsiness, and headache have each been reported in about one in fifty patients treated with GLUCOTROL. They are usually transient and seldom require discontinuance of therapy.

**Laboratory Tests:** The pattern of laboratory test abnormalities observed with GLUCOTROL was similar to that for other sulfonylureas. Occasional mild to moderate elevations of SGOT, LDH, alkaline phosphatase, BUN and creatinine were noted. One case of jaundice was reported. The relationship of these abnormalities to GLUCOTROL is uncertain, and they have rarely been associated with clinical symptoms.

## OVERDOSAGE

There is no well documented experience with GLUCOTROL overdosage. The acute oral toxicity was extremely low in all species tested ($LD_{50}$ greater than 4 g/kg).

## OVERDOSAGE *(continued)*

Overdosage of sulfonylureas including GLUCOTROL can produce hypoglycemia. Mild hypoglycemic symptoms without loss of consciousness or neurologic findings should be treated aggressively with oral glucose and adjustments in drug dosage and/or meal patterns. Close monitoring should continue until the physician is assured that the patient is out of danger. Severe hypoglycemic reactions with coma, seizure, or other neurological impairment occur infrequently, but constitute medical emergencies requiring immediate hospitalization. If hypoglycemic coma is diagnosed or suspected, the patient should be given a rapid intravenous injection of concentrated (50%) glucose solution. This should be followed by a continuous infusion of a more dilute (10%) glucose solution at a rate that will maintain the blood glucose at a level above 100 mg/dL. Patients should be closely monitored for a minimum of 24 to 48 hours since hypoglycemia may recur after apparent clinical recovery. Clearance of GLUCOTROL from plasma would be prolonged in persons with liver disease. Because of the extensive protein binding of GLUCOTROL, dialysis is unlikely to be of benefit.

## DOSAGE AND ADMINISTRATION

There is no fixed dosage regimen for the management of diabetes mellitus with GLUCOTROL or any other hypoglycemic agent. In addition to the usual monitoring of urinary glucose, the patient's blood glucose must also be monitored periodically to determine the minimum effective dose for the patient; to detect primary failure, i.e., inadequate lowering of blood glucose at the maximum recommended dose of medication; and to detect secondary failure, i.e., loss of an adequate blood-glucose-lowering response after an initial period of effectiveness. Glycosylated hemoglobin levels may also be of value in monitoring the patient's response to therapy.

Short-term administration of GLUCOTROL may be sufficient during periods of transient loss of control in patients usually controlled well on diet.

In general, GLUCOTROL should be given approximately 30 minutes before a meal to achieve the greatest reduction in postprandial hyperglycemia.

**Initial Dose:** The recommended starting dose is 5 mg, given before breakfast. Geriatric patients or those with liver disease may be started on 2.5 mg.

**Titration:** Dosage adjustments should ordinarily be in increments of 2.5-5 mg, as determined by blood glucose response. At least several days should elapse between titration steps. If response to a single dose is not satisfactory, dividing that dose may prove effective. The maximum recommended once daily dose is 15 mg. Doses above 15 mg should ordinarily be divided and given before meals of adequate caloric content. The maximum recommended total daily dose is 40 mg.

**Maintenance:** Some patients may be effectively controlled on a once-a-day regimen, while others show better response with divided dosing. Total daily doses above 15 mg should ordinarily be divided. Total daily doses above 30 mg have been safely given on a b.i.d. basis to long-term patients.

In elderly patients, debilitated or malnourished patients, and patients with impaired renal or hepatic function, the initial and maintenance dosing should be conservative to avoid hypoglycemic reactions (see PRECAUTIONS section).

**Patients Receiving Insulin:** As with other sulfonylurea-class hypoglycemics, many stable non-insulin-dependent diabetic patients receiving insulin may be safely placed on GLUCOTROL. When transferring patients from insulin to GLUCOTROL, the following general guidelines should be considered:

> For patients whose daily insulin requirement is 20 units or less, insulin may be discontinued and GLUCOTROL therapy may begin at usual dosages. Several days should elapse between GLUCOTROL titration steps.
>
> For patients whose daily insulin requirement is greater than 20 units, the insulin dose should be reduced by 50% and GLUCOTROL therapy may begin at usual dosages. Subsequent reductions in insulin dosage should depend on individual patient response. Several days should elapse between GLUCOTROL titration steps.

During the insulin withdrawal period, the patient should test urine samples for sugar and ketone bodies at least three times daily. Patients should be instructed to contact the prescriber immediately if these tests are abnormal. In some cases, especially when patient has been receiving greater than 40 units of insulin daily, it may be advisable to consider hospitalization during the transition period.

**Patients Receiving Other Oral Hypoglycemic Agents:** As with other sulfonylurea-class hypoglycemics, no transition period is necessary when transferring patients to GLUCOTROL. Patients should be observed carefully (1-2 weeks) for hypoglycemia when being transferred from longer half-life sulfonylureas (e.g., chlorpropamide) to GLUCOTROL due to potential overlapping of drug effect.

## HOW SUPPLIED

GLUCOTROL tablets are white, dye-free, scored, diamond-shaped, and imprinted as follows:

5 mg–Pfizer 411; 10 mg–Pfizer 412.

5 mg Bottles: 100's (NDC 0049-4110-66); (NDC 0662-4110-66); 500's (NDC 0049-4110-73); (NDC 0662-4110-73); Unit Dose 100's (NDC 0049-4110-41); (NDC 0662-4110-41)

10 mg Bottles: 100's (NDC 0049-4120-66); (NDC 0662-4120-66); 500's (NDC 0662-4120-73); Unit Dose 100's (NDC 0049-4120-41); (NDC 0662-4120-41)

**RECOMMENDED STORAGE:** Store below 86°F (30°C).

**CAUTION:** Federal law prohibits dispensing without prescription.

©1986 PFIZER INC.

65-4227-00-7

Printed in U.S.A.
Revised Dec. 1986

HV2332
L626 Lifestyle lessons: a
guide to diabetes care.

|  | DATE DUE |  |  |
|---|---|---|---|
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |

AMERICAN FOUNDATION FOR THE BLIND, INC.
15 WEST 16th STREET
NEW YORK, N. Y. 10011

Pfizer **Roerig**

RGX025X90 © 1990, Pfizer Inc. Printed in U.S.A. April, 1990